Focus on Sciences **Original Article**

May 2017, Volume 3, Issue 2

Effects of Cold Air on Cardiovascular Disease Risk Factors in Humans

Shuyu Zhang [1], Xiakun Zhang [2,*], Zhengzhong Kuang [3]

[1] Hebei Provincial Meteorological Bureau, Shijiazhuang, China
[2] National Meteorological Center, China Meteorological Administration, Beijing, China
[3] School of Applied Meteorology, Nanjing University of Information Sciences and Technology, Nanjing, China

** Corresponding author: Xiakun Zhang, National Meteorological Center of China Meteorological Administration, Beijing 100081, China. E-mail: zhangxk@cma.gov.cn*

DOI: 10.21859/focsci-03021402

Submitted: 12.28.2016
Accepted: 02.15.2017

Keywords:
Myoglobin
Blood Viscosity
Cardiac Troponin I

Abstract

Introduction: Zhangye, a city of the Province of Gansu, China was choose as the location for the experiment. After conducting health screening and blood tests, 30 patients with cardiovascular disease and 40 healthy persons were joined up as case and control groups, respectively. The cold air's effects on cardiovascular disease were studied by examining blood pressure and some biochemical indicators of them.

Methods: The experiment was carried out in the time of cold air exposure on April 27–28, 2013. Blood lipid, whole blood viscosity, cardiac troponin I, myoglobin (Mb), and endothelin-1 in all subjects were evaluated 24 h before (morning of April 26, 2013), during (temperature dropped to the lowest at 7:00 am–8:00 am, April 28, 2013), and 24 h after cold exposure. The variations in each biochemical indicator were analyzed before the cold exposure, during it, and after it.

Results: Results showed that the cold air exposure increased low and midst shear of whole blood viscosity and the low and high shear of whole blood reduced viscosity. This phenomenon led to excessive blood clotting and high aggregation and viscosity state in cardiovascular disease patients and healthy subjects. In addition, cold air exposure evidently increased serum triglyceride and especially lower density lipoprotein cholesterol and decreased higher density apolipoprotein and lipoprotein cholesterol. These indicators cause blood viscosity and increase in cholesterol and platelet granules, which are deposited in the vascular wall, thus further aggravating atherosclerosis. During cold air exposure, the concentration of vascular endothelin-1 significantly increased. Moreover, Mb and cardiac troponin I gradually increased and caused vasoconstriction and damage to myocardial cells. The main conclusions: The cold air can affect both patients with cardiovascular disease and people in healthy subjects. Firstly, cold air's effects can make excessive blood clotting and high aggregation and viscosity state and this can affect the blood rheology. Secondly, the cold air can promote the occurrence and development of atherosclerosis. Thirdly, the cold air can vasoconstriction and damage to myocardial cells.

Conclusions: When cold air transit formed by special meteorological conditions, the blood flow, blood lipid and myocardial serum markers were produced significant adverse effects, the cold air can cause disease of heart head blood-vessel crowd development.

INTRODUCTION

Cold air is one of key meteorological risk elements that affects the increasing in mortality and morbidity of cardiovascular and cerebro- vascular diseases [1-5]. Previous research [6] suggested that weather in winter half year or summer half year, in Changchun City, both the sharp changes in air pressure and temperature are prone to cause coronary heart disease relapse, the relatively huge rise of air pressure and fall of air temperature are unfavorable to cerebral hemorrhage and cerebral infarction, and the high blood pressure is mainly related to air pressure fluctuations and relatively humid weather. Acute cold stimulation can increase systolic blood pressure above 20 mmHg in humans [7-9], and the transient low temperature stimulation can rapidly increase blood pressure both in humans and animals [10-14]. Several epidemio-

logical studies demonstrated that low temperature during the cold season can result in hypertensive diseases [15] and rise the frequency of hypertension-associated cardiovascular diseases, for example, myocardial infarction and stroke [16-18]. Lipid metabolism disorder is a major risk factor for cardiovascular and cerebrovascular diseases [19]. One of the serum proteins, high-density lipoprotein cholesterol (HDL-C) has an anti-atherosclerosis effect, can reduce the risk of cardiovascular and cerebrovascular diseases [20]. A current research indicates that an increase in HDL-C level per 0.023 mmol/L decreases the coronary heart diseases' hazard by 2% for males and 3% for females [21]. Triglyceride (TG) has a different function with HDL-C. Increase in TG levels can cause "high blood viscosity." Viscous blood is caused by high lipid levels and increase cardiovascular and cerebrovascular disease risk [22]. LDL can initiate and maintain the inflammatory reaction of the blood vessel wall through oxidative damage to endothelial and smooth muscle cells to develop atherosclerosis [23]. Meanwhile, LDL induces excessive inflammatory cells to aggregate and aggravate inflammatory response by promoting the secretion of inflammatory mediators, thus aggravating atherosclerosis [24]. In addition, Apo-lipoprotein A1 (ApoA1) decrease and Apo-lipoprotein B (ApoB) increase can be used as risk predictors of cardiovascular and cerebrovascular diseases [25]. Myoglobin (Mb), troponin I, and endothelin-1 are serum reflection markers of myocardial injury. Mb is the only contractile protein in myocardial infarction and possesses high sensitivity and specificity for myocardial necrosis or injury. Although Mb has low content in the blood, small amount of myocardial necrosis rapidly increases its concentration [26]. Myocardial troponin I is a superior serum marker in terms of specificity and sensitivity for myocardial injury. It is also a gold standard for diagnosing acute myocardial infarction [27]. Rats exposed to cold air simulation show a change in blood lipid, triglyceride, serum total cholesterol, HDL-C, VLDL-C, and blood viscosity [26, 28]. Are there the similar results in human during the occurrence and development of cold air? This study investigated these results in human subjects exposed to cold air. A moderate strength cold air that occurred on April 2013 at Zhangye City was elected as the test case in this research. Zhangye, one of the cities in Gansu Province, China is located in northern China. There are climate difficult to pondering, variable weather, and big differences in temperature in this city. This location is the bottleneck for China northwest's cold air going southeast. In one year, averagely about 95% of the cold air influencing China goes along the Province of Gansu. Bad weather have big effects on the local people's lives and health, in particular, patients with cardiovascular and cerebrovascular diseases. During the experiment, blood lipid, whole blood viscosity, cardiac troponin I, Mb, and other biochemical indicators of cardiovascular and cerebrovascular disease were evaluated before, during, and after. We analyzed the variations in the indicators before cold exposure, also during it and after it. Moreover, we also explored the cold air' effects on cardiovascular and cerebrovascular diseases risk factors in human subjects.

METHODS

We choose the City of Zhangye which is located in 38.9°N, 100.5°E as the study area. This is a city in Gansu Province, China and its varied weather and clean air reaches the air standards. West and northwest cold air with temperature variations pouring south passes through this location. A random cluster sampling method was used in this experiment. A monitoring point was set up in the People's Hospital of Zhangye City. Forty-to-70-year-old residents' health records within one kilometer from that hospital were checked. Patients with cardiovascular and cerebrovascular diseases and without organic diseases were choose based on blood test and health screening. Before the formal research, 70 volunteer patients with cardiovascular disease who have no alcohol and drug influences in last 3 days were distributed to the patient group. Meanwhile, 70 healthy subjects, who are physically and mentally sound and have not had any disease recently, were selected as the control group. Volunteers went to the People's Hospital of Zhangye City every morning from April 26–29, 2013. A questionnaire surveying physical conditions, diet, medication, and activities, among others, was administered to the study groups. The questionnaire aimed to get out confounding factors, so that we could ensure the same exposure history between the patient and control groups. Non-compliance (absent or be latein blood measurements) and mismatch conditions (any drug-taking, mental stimulation, getting influenza or any other diseases) would be excluded. Totally 30 patients (16 males and 14 females) with cardiovascular or cerebrovascular disease and 40 healthy controls (24 males and 16 females) strictly completed the experiment and were included in continuous analysis. Cardiovascular and cerebrovascular diseases include thrombosis, stroke, myocardial infarction, coronary heart disease, and high blood pressure.

Ethical Considerations for Human Subjects: The study was reviewed and approved by the Medical Ethics Committee of Zhangye City People's Hospital before the experiment began. All the volunteers provided their written informed consent to participate in this experiment. This consent procedure was approved by the Medical Ethics Committee of Zhangye City People's Hospital and all the written informed consent was archived by the Committee. Measurement indicators: Biochemical indicators include blood lipid (HDL-C, TC, TG, LDL-C, VLDL-C, ApoA1 and ApoB), whole blood viscosity, Mb, cardiac troponin I, and endothelin-1, among others. Sample collection: Samples of fasting venous blood (5 mL) were collected from each volunteer 24 h before (morning of April 26, 2013), during (temperature dropped to the lowest at 7:00 am–8:00 am, April 28, 2013), and 24 h after cold exposure (morning of April 29, 2013). Samples were collected in vacuum blood collection tubes without anticoagulant and then centrifuged at 3,000 rpm. Subsequently, the serum was frozen at −80 °C. Determination: The enzyme-linked immunosorbent assay (ELISA) double antibody sandwich method was used to measure biochemical indicators. A micro-titer plate was coated with purified antibody to produce a solid-phase antibody. A test sample and the enzyme reagent were then added to form an antibody–antigen–enzyme–antibody complex. After washing, a chromogenic agent was added, and the absorbance was measured at 450 nm before calculating the concentration of the test sample. The blood was collected by vacuum blood collection tube with sodium citrate and measured by the automatic blood rheometer (LGR80, Steellex, China) for the shear rate of the whole blood viscosity.The ELISA kit was obtained from an Ameri-

can R&D company and packaged by Xi'an Kehao Biological Engineering Co., Ltd. (Xi'an, China). The micro-plate reader was acquired from Tecan Company in Austria. Detection was performed by the Medical Research Center in Lanzhou University.

Meteorological data: The crowd experimental study in Zhangye City was conducted on April 27–28, 2013 during the onset of cold weather. The Lanzhou Central Meteorological Observatory provided cold air weather data, including temperature, atmospheric pressure, weather forecast for cold air activity, and other hourly monitoring data. Cold air weather event was determined according to China's Cold Air Level National Standard GB/T20484-2006 developed by the Central Meteorological Observatory in 2006 [13]. Statistical Methods: SPSS13.0 software was used for statistical data analysis. The chi-square test was carried out to compare the sex and age composition of the patient and control groups. A randomized block design two-factor variance analysis was employed for blood indicators (including the whole blood viscosity, whole blood reduced viscosity and plasma viscosity) of different times, groups, and gender. The Mann–Whitney U test was conducted to compare the differences between patient and control groups. Before, during and after the cold air exposure, the indicators (including the blood lipid and serum reflection markers of myocardial injury) were analyzed with a one way ANOVA and the comparison of patient and control groups were analyzed with non-parameters Wilcoxon symbols test. These test standards were based on $\alpha = 0.05$.

RESULTS

Analysis of Changes in Cold Air

Table 1 shows the minimum temperature of 16.2 °C and 8.8 °C on April 26 and 28, respectively. The minimum temperature dropped by 7.4 °C in 48 h. China's national cold air level standards (GB/T20484-2006) confirmed that cold air showing a daily minimum temperature drop greater than or equal to 6 °C but less than 8 °C is considered a moderate strength cold air weather event. This cold air weather event influenced Zhangye temperature at 6:00 am on 27 April. Minimum temperature was achieved at 7:00 am on April 28, and cold air activity ended at 23:00 pm on the same day.

Analysis of the Basic Situation of the Experimental Crowd

The basic descriptions of the subjects participating in the moderate-intensity cold experiment conducted on April 26–29 are as follows (Table 2). The patient group comprised 30 cases, with a sex ratio of 1:1 and an average age of 59 years. This group consists of 6 cases of cerebral thrombosis, 2 cases of cerebral hemorrhage, 12 cases of coronary heart disease, and 10 cases of hypertension. The control group comprised 40 cases, with a sex ratio of 3:2 and an average age of 55 years. Difference in sex and age composition between the patient and control groups was not statistically significant ($P > 0.05$).

Analysis of Changes in Whole Blood Viscosity

Table 3 shows the comparative analysis of changes in whole blood viscosity before and during cold air exposure. The low, mid, and high shear of whole blood viscosity in the patient group increased by 1.4, 0.9, and 0.0 mPa.S compared with those before cold air exposure, and those in the control group increased by 3.6, 1.3, and 0.0 mPa.S, respectively. Low and mid shear of whole blood viscosity in the patient and control groups were significantly higher than those before cold air exposure ($P < 0.05$), but the high shear had no evident change. Regardless of gender, the low, mid, and high shear of whole blood viscosity in the patient group had no significant difference between before and during cold exposures. However, in the control group, low shear in female was higher than that in male and mid shear in male was higher than that in female. Significant difference was not observed between male and female in other indicators. A comparison between the patient and control groups before cold air exposure showed that the low and mid shear of whole blood viscosity in the patient group were significantly higher than those in the control group ($P < 0.05$). During cold air exposure, the low shear of whole blood viscosity in the control group was significantly higher than that in the patient group ($P < 0.05$). Significant difference was not observed between the other indicators of the two groups.

Table 1: Basic Meteorological Data of the Cold Air Event in Zhangye City on April 2013 (°C)

Variables	26th	27th	28th	29th
$Tmax_{24}$	26.1	19.4	16.4	26.5
$Tmin_{24}$	16.2	14.9	8.8	10.4
$\Delta Tmin_{48}$		7.4	4.5	

$Tmax_{24}$ denotes the daily maximum temperature, $Tmin_{24}$ denotes the daily minimum temperature, and $\Delta Tmin_{48}$ denotes the minimum temperature difference in 48 h.

Table 2: Gender and Age Compositions of the Patient and Control Groups

Group	Cases	Gender/n (%)		Age Composition/n (%)			
		Male	Female	40-	50-	60–70	
Control	40	24 (60.0)	16 (40.0)	11 (27.5)	14 (35.0)	15 (37.5)	55 ± 9.8
Patient	30	15 (50.0)	15 (50.0)	9 (30.0)	9 (30.0)	12 (40.0)	59 ± 10.0
Total	70	39 (57.1)	31 (42.9)	20 (28.6)	23 (32.8)	27 (38.6)	57 ± 9.6

Table 3: Comparison of Whole Blood Viscosity (mPa.S) before and During Cold Air Exposure

Gender	Low Shear		Midst Shear		High Shear	
	Before	During	Before	During	Before	During
Patient						
Male	10.7 ± 2.0	12.1 ± 1.4*	6.5 ± 1.1	7.4 ± 1.1*	5.6 ± 0.6	5.6 ± 0.8
Female	10.7 ± 1.8	12.0 ± 1.9*	5.6 ± 1.0	6.5 ± 0.9*	5.2 ± 1.0	5.2 ± 0.6
Total	10.7 ± 1.9#	12.1 ± 1.7*	6.1 ± 1.1#	7.0 ± 1.0*	5.4 ± 0.8	5.4 ± 0.7
Control						
Male	9.1 ± 1.8	12.1 ± 1.5*	5.1 ± 0.8	6.8 ± 1.1*	5.8 ± 0.7	5.7 ± 0.7
Female	9.3 ± 1.7	13.4 ± 2.2*	5.4 ± 1.0	6.4 ± 0.9*	5.4 ± 0.9	5.6 ± 0.9
Total	9.2 ± 1.8	12.8 ± 1.9*#	5.3 ± 0.9	6.6 ± 1.0*	5.6 ± 0.8	5.6 ± 0.8

* Compared with the indicators before cold air exposure, P < 0.05; # compared patient and control groups at the same time, P < 0.05

Table 4: Comparison between Whole Blood Reduced Viscosity and Plasma Viscosity (mPa.S) before and During Cold Air Exposure

Gender	Low Shear of Whole Blood Reduced Viscosity		High Shear of Whole Blood Reduced Viscosity		Plasma Viscosity	
	Before	During	Before	During	Before	During
Patient						
Male	20.7 ± 2.3	22.7 ± 1.3*	7.4 ± 1.2	8.4 ± 2.2*	1.7 ± 0.2	1.4 ± 0.1*
Female	21.5 ± 2.9	22.9 ± 2.1*	7.4 ± 1.0	8.2 ± 1.0*	1.3 ± 0.1	1.3 ± 0.1
Total	21.1 ± 2.6	22.8 ± 1.7*#	7.4 ± 1.1	8.3 ± 1.6*#	1.5 ± 0.2	1.4 ± 0.1
Control						
Male	21.3 ± 2.3	22.1 ± 2.0*	7.9 ± 1.0	8.1 ± 1.0	1.4 ± 0.1	1.4 ± 0.1
Female	20.8 ± 2.0	21.9 ± 2.3*	7.2 ± 1.1	8.2 ± 1.1*	1.4 ± 0.2	1.4 ± 0.2
Total	21.1 ± 2.2	22.0 ± 2.2*	7.6 ± 1.1	8.2 ± 1.1	1.4 ± 0.2	1.4 ± 0.2

* Compared with the indicators before cold air exposure, P < 0.05; # compared patient and control groups at the same time, P < 0.0

Table 5: Level of Blood Lipid during Cold Exposure

Indicators	Patient Group			Control Group		
	Before	During	After	Before	During	After
TC	5.12 ± 1.31	5.35 ± 0.91	5.01 ± 1.25	4.95 ± 0.82	5.01 ± 0.84	4.89 ± 0.90
TG	1.91 ± 1.42	2.25 ± 1.36*	2.02 ± 1.32	2.21 ± 1.33	2.41 ± 1.68	2.71 ± 3.41*
HDL-C	1.42 ± 0.33	1.39 ± 0.34	1.35 ± 0.31*	1.33 ± 0.37	1.31 ± 0.40	1.29 ± 0.25
LDL-C	2.84 ± 0.98	2.99 ± 0.60	2.89 ± 0.87	2.58 ± 0.86	2.65 ± 0.89	2.59 ± 0.76
VLDL-C	0.86 ± 0.61	1.02 ± 0.64*	0.97 ± 0.65	0.99 ± 0.54	1.08 ± 0.76	1.01 ± 0.55
ApoA1	1.51 ± 0.20	1.46 ± 0.23*	1.50 ± 0.31	1.36 ± 0.16	1.28 ± 0.26*	1.31 ± 0.18
ApoB	0.98 ± 0.20	1.04 ± 0.23	1.01 ± 0.18	1.06 ± 0.17	1.11 ± 0.16	1.08 ± 0.19

* Compared with the indicators before cold air exposure, P < 0.05

Table 4 shows the comparison of whole blood reduced viscosity between during and before cold air exposure. The low and high shear in the patient group increased by 1.7 and 0.9 mPa.S, respectively, and those in control group increased by 0.9 and 1.3 mPa.S, respectively. Low and high shear in the patient group were significantly higher than those before cold air exposure (P < 0.05), and low shear in the control group was significantly higher than that before cold air exposure (P < 0.05); the total high shear had no significant change. However, the high shear in female in the control group was significantly higher than that before cold air exposure (P < 0.05). Before cold air exposure, plasma viscosity in the patient and control groups were 1.5 and 1.4 mPa.S, respectively. During cold exposure, plasma viscosity in both groups was 1.4 mPa.S and had no significant difference compared with before cold air exposure (P > 0.05). The high shear of male in the control group was significantly higher than that in the patient group (P < 0.05). No significant differences between other indicators were observed. A comparison between the patient and control groups at the same time showed that each indicator of the two groups had no significant difference before cold air exposure. Low and high shear of whole blood reduced viscosity in the patient group were significantly higher than those in the control group during cold air exposure (P < 0.05). As

shown in the above analysis, the low and mid shear of whole blood viscosity and the low and high shear of whole blood reduced viscosity in both groups significantly increased before and during cold air exposure. This result suggests that the cold air affected the blood rheology of cardiovascular and cerebrovascular patients and healthy controls in different degrees, changed the fluidity and viscosity of blood, as well as led to excessive blood clotting and high aggregation and viscosity state. Cold air was closely related to the occurrence and development of cardiovascular and cerebrovascular diseases. This finding indicates that the special meteorological conditions formed by cold air exposure have an effect on blood flow and are major risk factors that cause cardiovascular and cerebrovascular diseases.

Analysis of Blood Lipid Test Results

As shown in Table 5, sera TC, TG, LDL-C, VLDL-C, and ApoB levels in the patient group during cold air exposure were higher than those before cold air exposure and increased by 0.23, 0.34, 0.15, 0.16, and 0.06 mmol/L, respectively. However, HDL-C and ApoA1 levels decreased by 0.03 and 0.05 mmol/L, respectively. After cold air exposure, sera TC, TG, LDL-C, VLDL-C, and ApoB levels were decreased and all indicators were higher than those before cold air exposure except TC. HDL-C continued to decline and decreased by 0.04 mmol/L compared with before cold air exposure. ApoA1 slightly increased but remained lower than that before cold air exposure. Sera TC, TG, LDL-C, VLDL-C, and ApoB levels in the control group increased by 0.06, 0.02, 0.07, 0.09, and 0.05 mmol/L, respectively, and were higher during cold air exposure compared with those before exposure. However, HDL-C and ApoA1 levels decreased by 0.02 and 0.08 mmol/L, respectively. After cold air exposure, all seven blood lipid indicators in the control group had similar changes as those in the patient group. The TG, VLDL-C, and ApoA1 during cold air exposure and the HDL-C after cold air exposure in the patient group had significant difference compared with those before cold air exposure. Blood lipid indicator changes in the patient and control groups had no significant difference. These results show that cold air had significantly affected sera HDL-C, ApoA1, TG, and VLDL-C levels. Thus, the effects of cold air on HDL-C, ApoA1, TG, and VLDL-C may be considered among the major risk factors causing cardiovascular and cerebrovascular diseases.

Analysis of the Serum Reflection Markers of Myocardial Injury Test Results

As shown in Table 6, the average level of Mb in both groups significantly changed ($P < 0.05$) before, during, and after cold air exposure and showed a positive increasing trend. Compared with before cold air exposure, the Mb in the patient group during and after cold air exposure increased by 124.5 and 644.1 ng/L, respectively, and the Mb in the control group during and after cold air exposure increased by 163.2 and 768.3 ng/L, respectively. Compared with during cold air exposure, the Mb in the patient group after cold air exposure increased by 519.6 ng/L and the Mb in the control group after cold air exposure increased by 605.1 ng/L. The Mb in both groups after cold air exposure had significant differences ($P < 0.05$) compared with those before and after cold air exposure. However, significant difference was not observed ($P > 0.05$) between the Mb in both groups during the same period of the cold air activities.

As shown in Table 6, the average level of troponin I in both groups slightly increased before and during cold air exposure but showed no significant differences ($P > 0.05$). Compared with before cold air exposure, troponin I in the patient and control groups during exposure increased by 1.2 and 4.0 ng/L, respectively. Troponin I in the patient group after cold air exposure evidently increased compared with before and during the exposure, which increased by 306.5 and 305.3 ng/L, respectively. Troponin I in the control group after cold air exposure also increased compared with before and during exposure, which increased by 197.1 and 193.1 ng/L, respectively. Troponin I in the patient group after cold air exposure exhibited significant differences ($P < 0.05$) compared with before and during exposure. However, troponin I in the control group after cold air exposure had no significant differences ($P > 0.05$) compared with before and during exposure. Significant difference was not observed ($P > 0.05$) between troponin I in the patient and control groups during the same period of the cold air activities. As shown in Table 6, the average level of endothelin-1 in each group before, during, and after cold air exposure significantly changed ($P < 0.05$). Compared with before cold air exposure, endothelin-1 during exposure appeared to have a positive growth in patient and control groups, which increased by 1.2 and 4.0 ng/L, respectively. Compared with before and after cold air exposure, endothelin-1 during exposure appeared to have a negative growth in the patient and control groups. The patient group decreased by 67.8 and 125.8 ng/L, and the control group decreased by 69.4 and 122.1 ng/L, respectively. Significant difference was not observed ($P > 0.05$) between

Table 6: Effects of Cold Air on Serum Reflection Markers of Myocardial Injury

Time	Patient Group (n = 30)			Control Group (n = 40)		
	Myoglobin	Troponin I	Endothelin-1	Myoglobin	Troponin I	Endothelin-1
Before	1567.4 ± 560.1	301.7 ± 117.6	139.7 ± 55.4	1439.4 ± 563.2	407.7 ± 207.3	159.8 ± 45.9
During	1691.9 ± 542.8*	302.9 ± 101.1	197.7 ± 86.7*	1602.6 ± 670.3	411.7 ± 213.9	212.5 ± 74.3
After	2211.5 ± 730.6*#	608.2 ± 309.3*#	71.9 ± 69.5*#	2207.7 ± 869.5*#	604.8 ± 327.1	90.4 ± 39.7*#

* Compared with the indicators before cold air exposure, $P < 0.05$; # compared with the indicators during cold air exposure, $P < 0.05$

endothelin-1 in the patient and control groups during the same period of the cold air activities. Results showed that Mb and troponin I appeared to continuously increase with the same trend appearing in both groups. The possible mechanism for this phenomenon is that the special meteorological conditions formed by the cold air activities, particularly the sudden change in temperature, because the corresponding stress reaction of the human body, increasing the load on the heart and brain blood supply as well as circulation and myocardial cell damage. This condition leads to increases in Mb and troponin I levels. Such damage caused by cold air exposure is difficult to recover. Endothelin-1 concentration in the patient and control groups increased during cold air exposure, decreased after cold air exposure, and was lower than that before exposure. Endothelin-1 levels in the patient group were statistically significant before, during, and after cold air exposure. However, no statistically significant difference was found between the endothelin-1 in the patient group before and during cold air exposure. This result indicates that the activity of cold air can affect changes in endothelin-1, thereby influencing the cardiovascular and cerebrovascular systems. In addition, the stress reaction of different groups (patient and control) were not the same under the climate conditions formed by cold air, making the corresponding indicators different. The effects of cold air on the three serum indicators in the patient group were more evident. This result indicates that the effects of cold air on cardiovascular and cerebrovascular patients are stronger than those on healthy controls, probably because patients are more sensitive to cold air. Each biochemical indicator changes during contact with cold air, which aggravates cardiovascular and cerebrovascular diseases in patients. In the same age phase as patients, some indicators of healthy subjects do not significantly change because healthy bodies have strong adaptability toward changes in the environment.

DISCUSSION

(1) With the effects of cold air, low and mid shear of whole blood viscosity and low and high shear of whole blood reduced viscosity in cardiovascular and cerebrovascular patients and healthy controls significantly increased. The increase in these parameters led to excessive blood clotting and high aggregation and viscosity state. This result shows that the special meteorological conditions formed by cold air have an effect on blood flow and these indicators may be the major risk factors that are the extent of reaction of blood viscosity and cause cardiovascular and cerebrovascular diseases.

(2) Sera TC, TG, LDL-C, VLDL-C, and ApoB levels in cardiovascular and cerebrovascular patients and healthy controls appeared to have an increasing trend as effects of cold air, whereas HDL-C and ApoA1 appeared to have a decreasing trend. Sera TG and VLDL-C of cardiovascular and cerebrovascular patients were significantly increased, whereas HDL-C and ApoA1 were significantly decreased. Only TG was significantly increased in healthy controls, whereas ApoA1 was significantly decreased. Thus, the effects of cold air were more evident in cardiovascular and cerebrovascular patients than those in healthy controls. An increase in TG leads to blood viscosity and a significantly decreased HDL-C and increased VLDL-C resulted in further aggravating atherosclerosis. Scriven et al. (1984), pointed out that a decrease in ApoA1 and an increase in ApoB can increase the risk of cardiovascular and cerebrovascular diseases [17]. Therefore, these indicators may be the major risk factors that are the extent of reaction of arterial vessel atherosclerosis and cause cardiovascular and cerebrovascular diseases.

(3) Mb and troponin I levels appeared to continuously increase both during and after cold air exposure and this showed that the cold air had a lag influence on them. Endothelin-1 concentration in patients and health controls evidently increased during cold air exposure, decreased after cold air exposure. Increased endothelin-1 concentration causes arterial blood vessels to shrink rapidly, thus leading to cardiovascular and cerebrovascular infarction. Continuous increase in Mb and troponin I levels could cause myocardial cell damage. In conclusion, these three biochemical indicators may be the major risk factors that are the extent of reaction of arterial vessel narrowing and myocardial cell damage and cause cardiovascular and cerebrovascular diseases.

ACKNOWLEDGMENTS

This work was supported by the National Natural Science Foundation of China (Grant Nos. 40975069 and 41375121).

AUTHORS' CONTRIBUTIONS

Shuyu Zhang, Xiakun Zhang and Zhengzhong Kuang participated in study design, clinical examinations and writing the article.

CONFLICTS OF INTEREST

The authors declare that the study don't have any competing interest.

FUNDING

there is no funding/support for this study.

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