

Effects of Cold Air on Cardiovascular Disease Risk Factors in Rat

Abstract

The purpose of this study is to explore possible implicit counteraccusations of cold air in cardiovascular complaint (CVD) threat in rats. Healthy Wistar rats were exposed to artificial cold air under laboratory conditions, and their systolic blood pressure, heart rate, vasoconstriction, CVD threat factors, and myocardial damage pointers after cold air exposure were determined and estimated. Systolic blood pressure, whole blood density, and tube position of norepinephrine, angiotensinII, low viscosity lipoprotein, total cholesterol, and fibrinogen in treatment groups increased significantly compared with control groups. No significant variations were set up in tube Mb and cTnT and myocardial towel between the treatment and control groups.

Keywords: cold air exposure • CVD threat factors • myocardial injury pointers

Introduction

Cold stress contributes to elevations in blood pressure. Studies report that a unforeseen exposure to the cold wave can increase the blood pressure of humans by 20 mmHg, especially when there exists cold wave convinced pain at the same time(2). A short period of patient low temperature stress could also lead to blood pressure elevation in both mortal and beast subjects. Several epidemiological studies have indicated that blood pressure is permanently elevated during the cold season. Habitual cold wave stress, generally lasting for further than three weeks, could also beget cold- convinced hypertension in rats. These cold- convinced blood pressure elevations have been proven to be related to the increase in norepinephrine (NE), epinephrine (EPI), and angiotensin II (ANGII) attention in the blood after cold stress. As critical vasoconstrictors, NE, EPI, and ANG II are essential factors of the sympathetic nervous system (SNS) and the rennin- angiotensin system (RAS), the activation of which could lead to elevations in blood pressure [1]. Acute cold wave stress accelerates the heart rate (HR), whereas a long period of cold stress could decelerate it down or make no change at all. To date, known studies substantially concentrate on the impact of constant low temperatures; the impacts brought about by the process of dwindling temperature in a natural cold air on HR remain unclear, particularly when the temperature changes from time to time. This observation could be used to explain the increase in CVD events in cold rainfall. Grounded on this knowledge, we hypothecated that the temperature drop process during exposure to cold air could also increase CVD threat factors. Epidemiological studies suggest a delayed peak in CVD events after a cold air. Similar events may be connected to the impact of the cold wave on CVD threat factors, similar as blood pressure, whole blood density (WBV), blood fibrinogen (FG) and lipids, among others [2]. Accoutrements and styles a cold air generally occurs in the downtime and early spring. It's a period of time in which the ambient temperature drops significantly. In China, cold charade are graded through the range of drop in the diurnal minimum temperature over 24 h, 48 h, or 72h. also, the range of drop in temperature from the former day's minimum temperature to another minimal temperature 24 h, 48 h,

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or 72 h latterly. This cold air was graded as a moderate cold air, with a drop in diurnal minimal temperature ranging from 6 °C to 8 °C according to the cold air grade standard of China. Details are shown in Figure 1. Over 7 time of collection of cold air experimental data in Zhangye City, the range of cold air temperatures used in this study passed about 103 times, or nearly 80 of all cold air in the area; hence, it could be regarded as a constantly being cold air in Zhangye City [3].

Results

Throughout the entire period of controlled casing and cold exposure, all groups of rats showed a stable growth rate and no significant difference was set up among them; no significant difference was observed either among the different groups in their HR. This suggests that the temperature dropping process of the cold air used in this study has no impact on the growth and heart rates still, compared with the control groups, a pronounced increase in SBP was set up in TD and TE after cold air exposure; no significant difference was observed between TDC and TEC.

Discussion

During the entire cold air process, the temperature dropped for about 8.9 °C within 51 h, with an average drop rate of 0.17 °C/h. This slow temperature change rate may have convinced adaption of the rats to the environmental conditions to some degree so as not to produce any impact on their HR. The HR findings may also be explained by the unchanged tube position of EPI through the entire temperature change process, as it frequently acts as a positive controller of HR. still, the cold air convinced SBP to increase, not only in TD but also in TE. This finding may be explained by the increased tube position of NE and ANG II in TD and TE. As critical factors of SNS and RAS, increased tube NE and ANG II could contribute to vasoconstriction and lead to the elevation of SBP [4]. Thus, the cold air may have actuated SNS and RAS and convinced SBP elevation in

the rats, analogous to the medium by which constant low temperature stresses induce SBP elevation. As an fluently observable and sensitive indicator of the cardiovascular system towards the cold wave, SBP has been reported to be more sensitive than the diastolic pressure in resting the circumstance of CVD events. Constantly elevated blood pressure could increase myocardial cargo and oxygen prostration and reduce blood inflow to the brain, which could ultimately induce myocardial and cerebral infarctions

Conclusions

In conclusion advanced situations of SBP, WBV and LDL/ HDL, total cholesterol (TC), and FG in blood may indicate advanced CVD pitfalls during cold air exposure; cold charade may ply nonstop impacts on SBP and other CVD threat factors [5]

Acknowledgement

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Conflict of Interest

No conflict of interest

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