Heart and Vessels (2019) 34:1309–1316
https://doi.org/10.1007/s00380-019-01355-8

ORIGINAL ARTICLE

Effect of acute aerobic exercise on arterial stiffness and thyroid-stimulating hormone in subclinical hypothyroidism

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Received: 19 September 2018 / Accepted: 1 February 2019 / Published online: 6 February 2019

Abstract

Acute exercise has been reported to increase thyroid hormone levels and decrease arterial stiffness in healthy young subjects. However, the effect of acute aerobic exercise on circulating thyroid hormone levels and arterial stiffness in patients with subclinical hypothyroidism remains unclear. The aim of this study was to investigate the effects of acute aerobic exercise on arterial stiffness and thyroid hormone levels, and any relationship between these endpoints, in patients with subclinical hypothyroidism. We studied patients with untreated subclinical hypothyroidism ($n = 53$, 65 ± 12 years old) compared with euthyroid subjects ($n = 55$, 64 ± 10 years old). Exercise analysis was performed with a ramp cycle ergometer test. Arterial stiffness (cardio-ankle vascular index, CAVI) was measured at baseline and 5 min after exercise. We collected participant blood samples for serum thyroid-stimulating hormone (TSH) and free thyroxine (FT4) measurements before and 5 min after exercise. The CAVI and serum TSH levels significantly decreased after exercise in the subclinical hypothyroidism group (CAVI; 8.1 ± 1.6 vs. 8.5 ± 1.5, $p < 0.001$, TSH; 6.7 ± 1.4 vs. 7.6 ± 1.2 μIU/ml, $p < 0.001$) and euthyroid group (CAVI; 7.6 ± 1.0 vs. 8.3 ± 0.9, $p < 0.001$, TSH; 2.2 ± 1.1 vs. 2.4 ± 1.2 μIU/ml, $p = 0.005$). The changes in CAVI from baseline compared with after exercise were lower, in absolute values, in the subclinical hypothyroidism group than in the euthyroid group (subclinical hypothyroidism group vs euthyroid group; ΔCAVI: -0.4 ± 0.6 vs. -0.7 ± 0.7, $p = 0.012$). The changes in serum TSH from baseline to after exercise were higher, in absolute values, in the subclinical hypothyroidism group than in the euthyroid group (subclinical hypothyroidism group vs euthyroid group; Δ serum TSH: -1.3 ± 1.4 vs. -0.3 ± 0.5, $p < 0.001$). The changes in CAVI from baseline to after exercise were negatively correlated with changes in TSH ($r = -0.32$, $p = 0.038$) in the subclinical hypothyroidism group. In conclusion, acute aerobic exercise decreased both arterial stiffness and serum TSH levels in patients with subclinical hypothyroidism and euthyroid subjects. While the absolute change in arterial stiffness decreased, the absolute change in serum TSH levels increased in patients with subclinical hypothyroidism compared with euthyroid subjects. These data suggest that subclinical hypothyroidism reduces CAVI during acute aerobic exercise. Further changes in absolute levels of serum TSH in subclinical hypothyroidism may result in reduced CAVI improvement by acute aerobic exercise.

Keywords Acute aerobic exercise · Thyroid-stimulating hormone (TSH) · Subclinical hypothyroidism · Arterial stiffness · Cardio-ankle vascular index (CAVI)

Introduction

Subclinical hypothyroidism is defined as an elevated thyroid-stimulating hormone (TSH) level in the context of normal free thyroxine (FT4) concentrations. It has been reported that subclinical hypothyroidism often lead to atherosclerosis, ischemic heart disease and cardiovascular mortality and morbidity [1, 2]. The mechanisms by which subclinical hypothyroidism causes arteriosclerosis include dyslipidemia, chronic inflammation, oxidative stress, insulin resistance, and decline in nitric oxide (NO) [3]. Nitric oxide reduction

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causes vascular endothelial dysfunction and increases arterial stiffness [4]. It is known that acute exercise increases circulating thyroid hormone levels [5, 6]. For example, Sullo et al. [6] found that rat serum TSH levels were increased at the end of exercise. In a human study, Ciloglu et al. [5] reported that high intensity acute aerobic exercise increased serum TSH levels and decreased free T3 in healthy younger subjects. Acute aerobic exercise also led to increase NO production by an increased circulating blood volume adding shear stress to the arterial wall. As a result, acute aerobic exercise decreased arterial stiffness [7–9]. No previous reports have investigated the association between serum thyroid hormone levels and arterial stiffness during acute aerobic exercise in patients with subclinical hypothyroidism.

The purpose of this study was to determine the effects of acute aerobic exercise on arterial stiffness and thyroid hormone levels, and any relationship between these endpoints, in patients with subclinical hypothyroidism.

Subjects

We studied fifty-three patients with untreated subclinical hypothyroidism (65 ± 12 years old) compared with fifty-five euthyroid subjects (64 ± 10 years old), who presented to the outpatient clinic of the Hyogo College of Medicine between January 2010 and April 2018. Patients were included in the study if they were aged ≥ 20 years. Patients were excluded if they had angina pectoris or acute coronary heart disease, valvular heart disease, current or recent history of congestive heart failure, atrial fibrillation, overt hypothyroidism or hyperthyroidism, and renal dysfunction.

Blood chemistry

Among 108 enrolled subjects, we collected blood samples for blood chemistry measurements from 42 and 36 patients in the subclinical hypothyroid group and euthyroid group, respectively. Blood samples were taken between 0900 and 1100 AM and were immediately placed on ice and then centrifuged. Specimens were stored at − 80°C until analysis. We measured blood levels of hemoglobin A1c (HbA1c), C-reactive protein (CRP), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglyceride (TG), lipids ratio (TG/HDL-C) and uric acid (UA) at baseline. Serum TSH (thyroid-stimulating hormone), FT3 (Free triiodothyronine) and FT4 (Free thyroxine) were measured before and after exercise. All samples were measured using the manufacturer's standard kits (Cobas® 8000, Roche Diagnostics, Mannheim, Germany). The euthyroid group and subclinical hypothyroidism group had patients with TSH levels of 0.5–4.4 μIU/ml and > 4.5 μIU/ml, respectively. Both groups had FT4 levels of 0.9–1.7 ng/dl.

Exercise studies

Exercise studies were performed in all 108 enrolled subjects using an upright cycle ergometer (Well Bike BE-350, Fukuda Denshi Corporation Limited, Tokyo, Japan) while measuring blood pressure and electrocardiogram. After a two-minute rest on the ergometer, the ramp exercise test was started for a pedaling frequency of 60 rpm. All subjects were encouraged to exercise until they were unable to continue. Arterial stiffness (CAVI) was measured at baseline and 5 min after exercise testing. CAVI was calculated using the arterial stiffness constant beta and the Bramwell–Hill equation [10]. The detailed CAVI method was described in our previous report [11]. CAVI was measured using a VaSera-1500 (Fukuda Denshi Corporation Limited, Tokyo, Japan). Mean blood pressure was calculated from the formula: mean blood pressure = [(2 × diastolic blood pressure) + systolic blood pressure]/3.

Echocardiography

All subjects had resting transthoracic echocardiography measured using a Hitachi-Aloka Prosound F75 (Hitachi-Aloka Medical, Tokyo, Japan) with a 3.8-MHz transducer according to the recommendations of the American Society of Echocardiography [12]. We assessed left atrial and left ventricular dimension by measuring left ventricular end-diastolic diameter (LVDd) and left ventricular end-systolic diameter (LVDs). We performed peak early diastolic phase (E) and late diastolic phase (A) mitral inflow velocities, and the E/A ratio by the pulsed wave Doppler. Peak early diastolic phase (E') and late diastolic phase (A') mitral annulus velocities were measured at the septal annulus by tissue Doppler imaging [13].

Ethics

This study was approved by the institutional review board of the Hyogo College of Medicine and written informed consent was obtained from all subjects.

Statistical analysis

All continuous data are presented as mean [± standard deviation (SD)]. We compared values between the subclinical hypothyroidism and the euthyroid groups using an unpaired t test and Chi-square test. The Spearman rank correlation was used to examine the correlation between the change in CAVI or in TSH and clinical variables. Multivariate analysis was performed using stepwise regression analysis with forward elimination to identify independent factors associated with the changes in CAVI. Parameters with $F > 4.0$ were entered into the regression analysis as independent variables. Statistical analyzes were performed using JMP version 10.0.1 (SAS Institute, Incorporated, Cary, NC, USA). *p* values of less than 0.05 were considered statistically significant.

Results

Patient characteristics

In the baseline characteristics of study participants, we found no significant differences in age, sex, body mass index (BMI), waist circumference, metabolic syndrome, medications, alcohol intake, smokers, systolic blood pressure, diastolic blood pressure, glycosylated HbA1c, UA, Cre, HDL-C, LDL-C, TG, lipids ratio (TG/HDL-C), CRP, FT3, FT4 and CAVI between the subclinical hypothyroidism and euthyroid groups. There were significantly decreased LVDd, LVDs and *E*/*A* ratios and significantly increased serum TSH levels, heart rate, and *A*′ in the subclinical hypothyroidism group compared with the euthyroid group. The *E*′ and *E*/*E*′ showed no statistical differences between the two groups (Table 1).

Effects of acute aerobic exercise on CAVI and circulating thyroid hormones

Acute aerobic exercise significantly decreased CAVI, FT3 and TSH levels and increased heart rate and mean blood pressure in the two groups. The subclinical hypothyroidism group exhibited increased systolic and diastolic blood pressure after exercise (Table 2).

Correlations between changes in CAVI, thyroid-stimulating hormone, heart rate and *A*′

Changes in the absolute values of CAVI were significantly decreased after exercise, whereas changes in the absolute values of serum TSH were significantly increased after exercise in the subclinical hypothyroid group compared with euthyroid group (Fig. 1). In the subclinical hypothyroidism group, changes in CAVI were significantly associated with changes in TSH (Fig. 2) and changes in heart rate (Fig. 3), but not with age and mean blood pressure. The changes in serum TSH levels were correlated with late diastolic mitral annular velocities in the subclinical hypothyroidism group (Fig. 4). The changes in serum TSH levels during exercise were significant negatively correlated with baseline serum TSH levels in patients with subclinical hypothyroidism ($r = -0.58$, $p < 0.001$), but not in euthyroid subjects.

Table 1 Baseline subject characteristics

	Euthyroid	Subclinical hypothyroidism
Age (years)	64 ± 10	65 ± 12
Sex (female/male)	19/36	18/35
Body mass index	23 ± 3	24 ± 4
Waist circumference (cm)	87.0 ± 9.8	87.3 ± 10.8
Metabolic syndrome, *n* (%)	12 (22)	16 (30)
Systolic blood pressure (mmHg)	125 ± 17	131 ± 18
Diastolic blood pressure (mmHg)	79 ± 11	83 ± 11
Heart rate (bpm)	62 ± 10	68 ± 11*
CAVI	8.3 ± 0.9	8.5 ± 1.5
Alcohol intake, *n* (%)	17 (31)	14 (26)
Smokers, *n* (%)	19 (35)	11 (21)
Hypertension, *n* (%)	18 (33)	18 (34)
Hyperlipidemia, *n* (%)	19 (35)	14 (26)
Diabetes, *n* (%)	5 (9)	5 (9)
Medication		
ARB, *n* (%)	7 (13)	4 (8)
CCB, *n* (%)	13 (24)	11 (21)
Statin, *n* (%)	13 (24)	7 (13)
Diabetes medication, *n* (%)	4 (7)	3 (6)
C-reactive protein (mg/dl)	0.1 ± 0.2	0.1 ± 0.3
HbA1c (%)	5.4 ± 0.5	5.9 ± 1.5
Uric acid (mg/dl)	5.5 ± 1.2	5.5 ± 1.3
Creatinine (mg/dl)	0.7 ± 0.1	0.8 ± 0.3
Free T3 (pg/dl)	2.9 ± 0.3	2.9 ± 0.4
Free T4 (ng/dl)	1.2 ± 0.2	1.2 ± 0.2
TSH (μIU/ml)	2.4 ± 1.2	7.6 ± 8.8*
HDL cholesterol (mg/dl)	60 ± 19	60 ± 19
LDL cholesterol (mg/dl)	121 ± 30	124 ± 30
TG (mg/dl)	117 ± 68	123 ± 78
TG/HDL cholesterol ratio	2.4 ± 2.1	2.4 ± 2.1
LVDd (mm)	47 ± 4	45 ± 4**
LVDs (mm)	29 ± 4	27 ± 3*
EF (%)	69 ± 5	70 ± 6
E wave	66 ± 15	62 ± 14
A wave	68 ± 16	72 ± 18
Dct (ms)	211 ± 45	207 ± 51
E/*A*	1.0 ± 0.4	0.9 ± 0.3*
E/*E*'	8.9 ± 2.4	9.0 ± 2.2
E′	7.7 ± 2.2	7.2 ± 2.0
A′	9.5 ± 1.7	10.1 ± 1.9*

CAVI cardio-ankle vascular index, *ARB* angiotensin type II receptor blocker, *CCB* calcium channel blocker, *HDL* cholesterol, high-density lipoprotein cholesterol, *LDL* cholesterol, low-density lipoprotein cholesterol, *TG* triglyceride, *LVDd* left ventricular end-diastolic diameter, *LVDs* left ventricular end-systolic diameter, *EF* left ventricular ejection fraction, *Dct* deceleration time

*$p < 0.05$, **$p < 0.01$

Table 2 Effects of acute aerobic exercise on CAVI and circulating thyroid hormones

	Euthyroid		Subclinical hypothyroidism	
	Before exercise	After exercise	Before exercise	After exercise
Systolic blood pressure (mmHg)	125 ± 17	131 ± 14	131 ± 18	135 ± 17**
Diastolic blood pressure (mmHg)	79 ± 11	80 ± 13	83 ± 11	85 ± 11*
Mean blood pressure (mmHg)	94 ± 12	97 ± 12*	98 ± 11	102 ± 12**
Heart rate (bpm)	62 ± 10	74 ± 13***	68 ± 11	78 ± 11***
CAVI	8.3 ± 0.9	7.6 ± 1.0***	8.5 ± 1.5	8.1 ± 1.6***
Free T3 (pg/dl)	2.9 ± 0.3	2.8 ± 0.3***	2.9 ± 0.4	2.7 ± 0.4***
Free T4 (ng/dl)	1.2 ± 0.2	1.2 ± 0.2	1.2 ± 0.2	1.2 ± 0.2
TSH (μIU/ml)	2.4 ± 1.2	2.2 ± 1.1**	7.6 ± 8.8	6.7 ± 9.0***

CAVI cardio-ankle vascular index

*$p<0.05$, **$p<0.01$, ***$p<0.001$

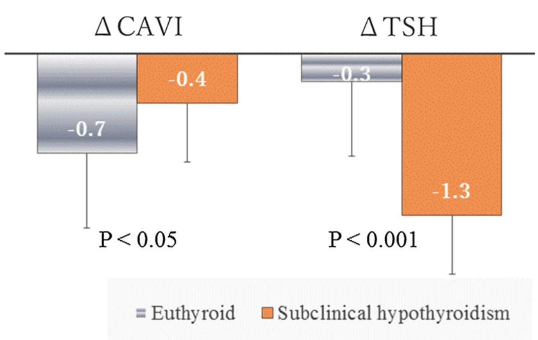

Fig. 1 Changes in CAVI and serum TSH levels before and after exercise. *ΔCAVI* changes in CAVI (arterial stiffness), *ΔTSH* changes in thyroid-stimulating hormone (μU/ml). Changes in the absolute values of CAVI were significantly decreased after exercise, whereas changes in the absolute values of serum TSH were significantly increased after exercise in the subclinical hypothyroid group compared with euthyroid group

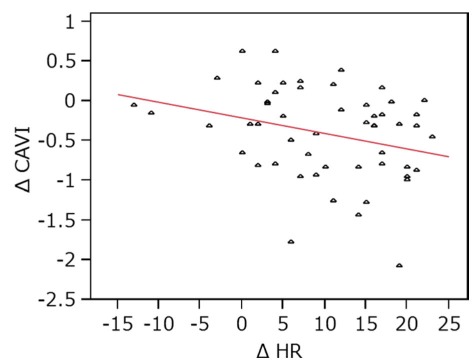

Fig. 3 Correlation between change in CAVI and change in heart rate in the subclinical hypothyroidism group. In the subclinical hypothyroidism group, change in CAVI was significantly associated with change in heart rate ($r = -0.30$, $p=0.03$, $n=53$). *ΔCAVI* changes in CAVI (arterial stiffness), *ΔHR* changes in heart rate (bpm)

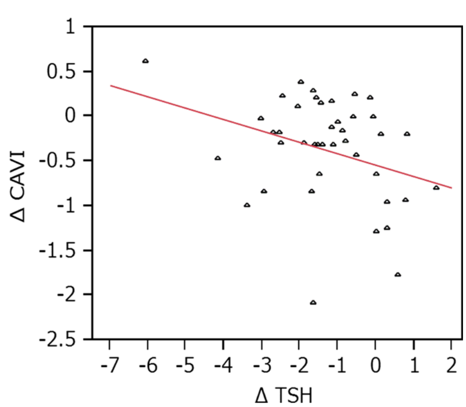

Fig. 2 Correlation between change in CAVI and change in circulating TSH in the subclinical hypothyroidism group. In the subclinical hypothyroidism group, change in CAVI was significantly associated with change in TSH ($r = -0.32$, $p=0.03$, $n=42$). *ΔCAVI* changes in CAVI (arterial stiffness), *ΔTSH* changes in thyroid-stimulating hormone (μU/ml)

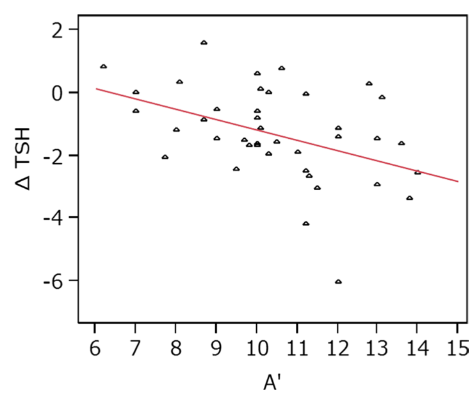

Fig. 4 Correlation between the change in TSH levels and A' at rest in the subclinical hypothyroidism group. In the subclinical hypothyroidism group, change in TSH was significantly associated with the peak velocity at the mitral annulus in late diastolic phase (A') during rest ($r = -0.43$, $p=0.01$, $n=40$). *ΔTSH* changes in thyroid-stimulating hormone (μU/ml), A' the mitral annulus in late diastolic phase (A') converted to absolute values

In the subclinical hypothyroidism group, changes in serum TSH levels, heart rate, mean blood pressure and A' were independently associated with changes in CAVI after stepwise multiple regression analysis (changes in serum TSH levels; regression coefficient = − 0.21; standard error = 2.40; F value = 9.53, changes in heart rate; regression coefficient = − 0.02; standard error =1.07; F value = 4.25, changes in mean blood pressure; regression coefficient =0.02; standard error = 1.25; F value = 4.97, A'; regression coefficient = −0.12.; standard error = 1.30; F value = 5.15). Non-accepted variables were age, sex and changes in free T3 in the subclinical hypothyroidism group.

Discussion

The main findings in the current study were: (1) CAVI (arterial stiffness) and serum TSH levels were significantly decreased after acute aerobic exercise in the subclinical hypothyroidism and euthyroid groups; and (2) The changes in CAVI by acute exercise tended to be greater in the euthyroid group compared with the subclinical hypothyroidism group. Additionally, changes in serum TSH levels by acute aerobic exercise were greater in the subclinical hypothyroidism group compared with the euthyroid group. (3) There was a negative correlation between exercise-induced changes in CAVI and exercise-induced changes in serum TSH levels in the subclinical hypothyroidism group.

Arterial stiffness and exercise

In the present study, CAVI was decreased after high intensity exercise in both groups. The changes in absolute values of CAVI after exercise were significantly lower in the subclinical hypothyroidism group compared with the euthyroid group. Several studies reported that acute aerobic exercise decreased arterial stiffness in healthy subjects [9, 14–17]. For example, Ashor et al. [14]demonstrated that aerobic exercise improved arterial stiffness. Additionally, higher intensity exercise was more effective at reducing arterial stiffness compared with lower intensity exercise. Wang et al. [17] and Yin et al. [18] found that 15 min of acute exercise resulted in a decrease in systemic arterial stiffness in healthy young men, with CAVI values of 6.8 ± 0.1 and 5.9 ± 0.1 at baseline and after exercise, respectively (mean reduction of approximately 0.9). In comparison, the current study found similar changes in CAVI during exercise in euthyroid subjects; however, baseline CAVI values were higher than those in previous studies. The different experimental results may be explained by the different ages of participants. Considered together, these findings suggest that the high intensity acute aerobic exercise may be beneficial by decreasing arterial stiffness. The present study found that changes in CAVI during exercise tended to be greater in the euthyroid group compared with the subclinical hypothyroidism group.

The possible mechanisms contributing to observed CAVI changes with exercise include endothelial dysfunction related to changes in arterial stiffness. In patients with subclinical hypothyroidism, increased diastolic blood pressure and arterial endothelial dysfunction resulting from a reduction in NO may be a major factor associated with exaggerated arterial stiffness. It was previously reported that increased shear stress during exercise leads to increased NO synthesis activity in healthy patients [19]. Conversely, subclinical hypothyroidism was associated with impaired endothelial vaso-relaxation due to reduced NO synthesis activity [20, 21]. Therefore, in this study, changes in the absolute values of CAVI after exercise due to endothelial dysfunction were likely to be smaller in the subclinical hypothyroidism group than in the euthyroid group.

Thyroid hormones and exercise

In our study, serum TSH levels were significantly decreased in the subclinical hypothyroid and euthyroid groups after exercise. Previous reports suggested that serum TSH levels decreased or increased after exercise. Animal studies found that exercise was associated with decreased serum thyroid hormone levels in sled dogs [22]. Krotkiewski et al. [23] demonstrated that serum TSH levels were reduced in obese women after exercise. It has been reported that serum leptin is associated with regulation of the hypothalamic–pituitary–thyroid axis. Fisher et al. [24] demonstrated that serum leptin levels decreased after acute exercise. Serum leptin levels are known to be correlated with serum TSH levels [25]. Hence, we speculate that decreasing serum TSH levels after exercise may contribute to exercise-induced changes in serum leptin levels. Future studies will be required to investigate the association between serum leptin and TSH levels during exercise.

In contrast, Hashimoto et al. [26] and Sullo et al. [6]found that serum TSH levels increased in healthy subjects and an animal model after physical exercise. We speculate that the mechanism by which TSH and FT3 levels decline after acute exercise may involve different factors. Sowers et al. [27] found that an exercise-induced elevation in glucocorticoids may lead to increased TSH levels and a significant reduction in FT3 levels. Ciloglu et al. [5] observed that changes to thyroid function depended upon the degree on exercise intensity. In acute aerobic exercise, serum TSH levels were higher during high intensity exercise (90% of maximum heart rate) compared with low intensity exercise (45% of maximum heart rate). The mechanism of increased serum TSH levels after high intensity exercise may involve exercise-induced elevated of thyroid hormones in peripheral tissues. Moreover, it is presumed that decreased serum thyroid hormone levels after exercise may prevent high thermogenesis during exercise [28]. In the current study, the reduced absolute values of serum TSH were higher in the subclinical hypothyroidism group than in the euthyroid group after exercise. In other words, the subclinical hypothyroidism group may exhibit an impaired ability to raise or maintain serum TSH levels after exercise, compared with the euthyroid group.

Subclinical hypothyroidism and diastolic dysfunction

The present study demonstrated that the changes in serum TSH levels were correlated with the late diastolic mitral annular velocities (A') in the subclinical hypothyroidism group.

Biondi et al. also found increased mitral doppler A wave velocities in patients with subclinical hypothyroidism [29, 30]. Previous studies suggested that left ventricular diastolic dysfunction involved reduced calcium reuptake into the sarcoplasmic reticulum through decreased expression of sarcoplasmic/endoplasmic reticulum Ca^{2+} ATPase (SERCA) and phospholamban activation in individuals with subclinical hypothyroidism [3, 31, 32].

Generally, early diastolic mitral annular velocities (E') and E/E' are widely used as indicators of left ventricular diastolic function using Doppler tissue imaging. However, in this study, E' or E/E' were not significantly different between the euthyroid and subclinical hypothyroid groups. The present study included very few cases where E/E' exceeds 15, suggesting the presence of mild left ventricular dysfunction. In mild left ventricular diastolic dysfunction, the elevation of A' reflects a compensatory increase of left atrial contraction to left ventricular filling pressure. Therefore, the subclinical hypothyroid group may have an increased A' due to mild left ventricular diastolic dysfunction compared with the euthyroid group.

Serum TSH levels and arterial stiffness

In this study, there was a negative correlation between changes in CAVI and changes in serum TSH levels during acute aerobic exercise in the subclinical hypothyroidism group. Moreover, the change in serum TSH level during exercise was significantly negatively correlated with baseline serum TSH levels. Our findings support previous studies, which indicated that serum TSH levels within the upper reference levels are associated with endothelial dysfunction [33]. In addition, Dardans et al. reported that TSH administration reduced endothelium-dependent NO [34]. However, it remains unclear why the impaired arterial stiffness during acute aerobic exercise was associated with decreased serum TSH levels. Further investigation of the association between the changes in CAVI or TSH levels during exercise is needed to clarify the clinical implications of CAVI in patients with subclinical hypothyroidism.

Limitations

There are several limitations in our study. First, this study was conducted in a single center with a small sample size. Second, we did not investigate thyroid hormone replacement therapy in the studied patients. Further exploration of whether the changes in arterial stiffening during exercise are reversed by thyroxine replacement therapy in subjects with subclinical hypothyroidism may be necessary. Third, this study included only short-term effects of exercise on arterial stiffness and future studies will be needed to assess long-term effects of exercise on arterial stiffness. Forth, we did not determine whether the change to arterial stiffness was associated with an increased risk for all-cause mortality.

Future directions

The present study found that subclinical hypothyroidism reduced arterial stiffening, as measured by CAVI, and that this reduction was impaired in subclinical hypothyroidism compared with euthyroidism. The acute effects of aerobic exercise on arterial stiffness may involve a decrease in serum TSH levels. The finding of impaired arterial stiffness during acute aerobic exercise may have important clinical implications for early arteriosclerosis in subclinical hypothyroidism subjects. Recently, Sato et al. [35] reported that subclinical hypothyroidism was associated with cardiac event rate and all-cause mortality, accompanied by exercise intolerance and pulmonary hypertension in patients with heart failure. However, to our knowledge, there is no evidence of a correlation between changes in serum TSH levels from acute exercise and arterial stiffness, cardiac event rate or mortality. Further investigation into the association between the changes in CAVI or serum TSH levels during the acute effects of aerobic exercise and cardiac event rate and mortality is needed to clarify the clinical implications of altered CAVI or serum TSH levels in subclinical hypothyroidism.

Conclusion

We investigated the acute effects of exercise on arterial stiffness (CAVI) in subclinical hypothyroidism and euthyroid groups. A significant reduction in CAVI during exercise was observed in both groups and this reduction was poor in the subclinical hypothyroidism group. These data suggest that subclinical hypothyroidism impairs CAVI during acute aerobic exercise. An increased change in absolute levels of serum TSH may limit the degree of CAVI improvement by acute aerobic exercise in subclinical hypothyroidism.

Acknowledgements We thank Ms. M Tanaka, S Makihara and C Misumi for their excellent technical assistance in the echocardiographic tracings.

Funding This work was supported by Grant-in-Aid for Researchers, Hyogo College of Medicine, 2018 and a contract research Grant, Public Interest Incorporated Association Japan Society of Ningen Dock, 2018. We thank Charles Allan, PhD, from Edanz Group (http://www.edanzediting.com/ac) for editing a draft of this manuscript.

Compliance with ethical standards

Conflict of interest The authors declare no conflicts of interest associated with this manuscript.

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