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ORIGINAL ARTICLE

Changes in arterial stiffness, carotid intima-media thickness, and epicardial fat after L-thyroxine replacement therapy in hypothyroidism☆

Abdel del Busto-Mesa[a], Julio Oscar Cabrera-Rego[b,*], Lisván Carrero-Fernández[b], Cristina Victoria Hernández-Roca[c], Jorge Luis González-Valdés[d], José Eduardo de la Rosa-Pazos[d]

[a] *Servicio de Endocrinología, Hospital Miguel Enríquez, La Habana, Cuba*
[b] *Unidad de Cuidados Coronarios Intensivos, Hospital Manuel Fajardo, La Habana, Cuba*
[c] *Departamento de Bioestadística, Hospital Miguel Enríquez, La Habana, Cuba*
[d] *Facultad de Medicina, Hospital Miguel Enríquez, La Habana, Cuba*

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KEYWORDS
Hypothyroidism;
Arterial stiffness;
Carotid intima-media thickness;
Epicardial fat;
Replacement therapy

Abstract
Objective: To assess the relationship between primary hypothyroidism and subclinical atherosclerosis and its potential changes with L-thyroxine replacement therapy.
Methods: A prospective cohort study including 101 patients with primary hypothyroidism and 101 euthyroid patients as controls was conducted from July 2011 to December 2013. Clinical, anthropometrical, biochemical, and ultrasonographic parameters were assessed at baseline and after one year of L-thyroxine replacement therapy.
Results: At baseline, hypothyroid patients had significantly greater values of blood pressure, total cholesterol, VLDL cholesterol, left ventricular mass, epicardial fat, and carotid intima-media thickness as compared to controls. Total cholesterol, VLDL cholesterol, ventricular diastolic function, epicardial fat, carotid intima-media thickness, carotid local pulse wave velocity, pressure strain elastic modulus, and β arterial stiffness index showed a significant and positive correlation with TSH levels. After one year of replacement therapy, patients with hypothyroidism showed changes in total cholesterol, VLDL cholesterol, TSH, carotid intima-media thickness, and arterial stiffness parameters.

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* Corresponding author.
E-mail address: jocabrera@infomed.sld.cu (J.O. Cabrera-Rego).

Conclusions: Primary hypothyroidism is characterized by an increased cardiovascular risk. In these patients, L-thyroxine replacement therapy for one year is related to decreased dyslipidemia and improvement in markers of subclinical carotid atherosclerosis.

PALABRAS CLAVE
Hipotiroidismo;
Rigidez arterial;
Grosor íntima-media carotídeo;
Grasa epicárdica;
Tratamiento sustitutivo

Modificaciones de la rigidez arterial, el grosor íntima-media carotídeo y la grasa epicárdica con tratamiento sustitutivo en el hipotiroidismo

Resumen
Objetivo: Determinar la relación del hipotiroidismo primario con la aterosclerosis carotídea subclínica y sus posibles modificaciones con la terapia sustitutiva.
Métodos: Se realizó un estudio de cohorte prospectivo que incluyó 101 pacientes con diagnóstico de hipotiroidismo primario y 101 pacientes eutiroideos como controles, desde julio del año 2011 hasta diciembre del 2013. Se incluyeron variables clínicas, antropométricas, bioquímicas y ultrasonográficas, evaluadas al inicio del estudio y al año de tratamiento sustitutivo con levotiroxina sódica.
Resultados: Los sujetos afectos de hipotiroidismo al inicio mostraron valores significativamente mayores de tensión arterial, colesterol total, colesterol VLDL, masa del ventrículo izquierdo, grasa epicárdica y grosor íntima-media carotídeo, respecto a los controles. El colesterol total, el colesterol VLDL, la función diastólica ventricular, la grasa epicárdica, el grosor íntima media, la velocidad de propagación del pulso carotídeo, la elastancia y el índice de rigidez arterial β mostraron una correlación positiva y significativa con las concentraciones de TSH. Al año de tratamiento, los pacientes afectos de hipotiroidismo tuvieron modificaciones para el colesterol total, el colesterol VLDL, la TSH, el grosor íntima-media y los parámetros de rigidez arterial.
Conclusiones: Los pacientes afectos de hipotiroidismo primario se caracterizan por mayor riesgo cardiometabólico. La sustitución con levotiroxina sódica en estos pacientes se relaciona con una mejoría de la dislipidemia, y mejoría de los indicadores de aterosclerosis carotídea subclínica al año de iniciado el tratamiento.

Introduction

Hypothyroidism results from deficient thyroid hormone secretion and is associated with comorbid conditions such as high blood pressure, dyslipidemia, and ischemic heart disease. Hypothyroidism is more prevalent between the third and sixth decades of life and in females. Its incidence in the general population ranges from 1% to 2%, and reaches 6–7% in females over 60 years of age. In Cuba, hypothyroidism is the second leading endocrine disease after diabetes mellitus.[1]

In patients with hypothyroidism increased total and LDL cholesterol levels and decreased HDL cholesterol levels have been found. It is thus clear that the condition involves an atherogenic profile, which may increase vascular risk in those who suffer from it.[2]

Cardiovascular risk appears to be independently associated with subclinical hypothyroidism in patients over 65 years of age. Women with hypothyroidism have a two-fold greater risk of developing atherosclerosis and a greater prior history of myocardial infarction as compared to controls. An increased incidence of peripheral artery disease, diastolic and systolic left ventricular dysfunction, and ischemic coronary events has also been reported in patients with subclinical hypothyroidism.[3]

It is currently known that cardiovascular changes in thyroid dysfunction are not restricted to clinically evident forms of dysfunction. There is considerable evidence to suggest that the cardiovascular system responds to the minimal but persistent changes in circulating thyroid hormone levels which are characteristic of subclinical thyroid dysfunction.[4]

Both clinical and subclinical hypothyroidism are related to a reversible state of endothelial dysfunction which may be responsible for high blood pressure and an increased risk of atherosclerosis. As TSH increases, flow-mediated vasodilation decreases, suggesting the presence of endothelial dysfunction. On the other hand, the cardiovascular change most commonly found in hypothyroidism is left ventricular (LV) diastolic dysfunction, characterized by slow myocardial relaxation and delayed ventricular filling.[5]

The increased risk of atherosclerosis in hypothyroidism mainly results from insulin resistance, and is linked to a low-grade chronic inflammation state promoted by the large quantity of adipokines secreted by adipose tissue, including tumor necrosis factor alpha and interleukin-6, which accelerate athrerosclerosis.[4]

Failure to adjust levothyroxine levels results in changes due to either excess or deficient drug dosages. However, progressive improvement of the clinical condition after