

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/41089343>

Dyslipidaemia and hypertension in patients with subclinical hypothyroidism

Article in *Prilozi / Makedonska akademija na naukite i umetnostite, Oddelenie za biološki i medicinski nauki = Contributions / Macedonian Academy of Sciences and Arts, Section of Biological and Medical Sciences* · December 2009

Source: PubMed

CITATIONS

30

READS

239

5 authors, including:



Velkoska Nakova Valentina

Goce Delcev University

43 PUBLICATIONS 314 CITATIONS

SEE PROFILE



Brankica Krstevska

Saints Cyril and Methodius University of Skopje

84 PUBLICATIONS 398 CITATIONS

SEE PROFILE



Marijan Bosevski

Saints Cyril and Methodius University of Skopje

314 PUBLICATIONS 12,727 CITATIONS

SEE PROFILE



Vladimir Serafimoski

Macedonian Academy of Sciences and Arts

35 PUBLICATIONS 245 CITATIONS

SEE PROFILE

DYSLIPIDAEMIA AND HYPERTENSION IN PATIENTS WITH SUBCLINICAL HYPOTHYROIDISM

Velkoska Nakova V.,¹ Krstevska B.,¹ Bosevski M.,²
Dimitrovski Ch.,¹ Serafimoski V.^{3,4}

¹*Endocrinology, Diabetes and Metabolic Disorders Clinic,
Medical Faculty, Skopje, R. Macedonia*

²*Cardiovascular Disease Clinic, Medical Faculty, Skopje, R. Macedonia*

³*Gastroenterohepatology Clinic, Medical Faculty, Skopje, R. Macedonia*

⁴*Macedonian Academy of Sciences and Arts, Skopje, R. Macedonia*

Abstract: *Objective.* The aim of this study was to assess whether subclinical hypothyroidism (SCH) is associated with dyslipidaemia and arterial hypertension.

Methods. At the Department of Endocrinology, Diabetes and Metabolic Disorders, Skopje, R. Macedonia, we examined 24 consecutive patients with SCH and 13 healthy controls in a period of 6 months. SCH was defined as an elevated thyrotropin (TSH) (> 4.2 mU/l) and normal free thyroxine (fT4) level (10.3–24.45 pmol/l). None of the patients had been previously treated with thyroxine. In all participants we determined blood pressure, body mass index (BMI), TSH, fT4, antibodies to thyroid peroxidase (TPOabs), total lipids (TL), total cholesterol (TH), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides.

Results. Mean diastolic blood pressure increased in SCH patients vis-a-vis controls (85 vs. 74 mmHg; $p < 0.05$). Mean values of TL, TH, HDL-C, LDL-C, triglycerides, TC/HDL-C, and LDL-C/HDL-C were no different in patients with SCH compared with controls. Individual analysis revealed that the percentages of patients with SCH having arterial hypertension (29%), hypertriglyceridaemia (34.78%), elevated LDL-C (41.66%), elevated TC/HDL-C (21.7%), and LDL-C/HDL-C (21.74%) ratios were higher than the percentages in controls. No significant correlation between TSH and biochemical parameters was detected.

Conclusion. Our study revealed that SCH patients have a greater prevalence of dyslipidaemia and arterial hypertension, and, as well, a greater value of mean diastolic pressure vs. control patients.

Key words. Subclinical hypothyroidism; arterial hypertension; dyslipidaemia; atherosclerosis; risk factors.

Introduction

SCH, also called mild hypothyroidism, is a term used for a condition in which there are small elevations in the thyroid-stimulating hormone, yet normal circulating levels of thyroid hormones. This condition is more common in the elderly and is found twice as often in women as in men [1]. In general, the prevalence of this condition in women ranges from 4% at age 20 to 17% at age 65 and in men from 2% at age 20 to 7% at age 65 [2, 3].

Thyroid hormones have significant effects on the synthesis, mobilization and metabolism of lipids [4].

Overt hypothyroidism is associated with significant increases in circulating concentrations of TH and LDL-C [5], but it is uncertain whether SCH is also associated with dyslipidaemia. Some case-control studies [6–9], but not others, have reported an association between SCH and dyslipidaemia in subjects with SCH compared with euthyroid controls. Two large cross-sectional studies [10, 11] reported increased serum TH, LDL-C, and triglycerides in subjects with SCH. By contrast several large cross-sectional studies found no significant difference in TH, LDL-C, and triglycerides between subjects with SCH and euthyroid subjects [12–17].

Whereas overt hypothyroid patients in the fifth and sixth decades of life have significantly higher diastolic blood pressure and arterial hypertension than age-matched controls, an association between arterial hypertension and SCH has not been reported [10, 18, 19].

Dyslipidaemia and arterial hypertension are risk factors for atherosclerosis. The idea that overt hypothyroidism promotes atherosclerosis has been generally accepted, but whether SCH is associated with increased risk of atherosclerosis, is still a matter of debate [20–22].

The study aimed to assess SCH effects on blood pressure and lipids.

Patients and methods

The study was conducted from 01.09. 2008 to 28.02. 2009, at the Department of Endocrinology, Diabetes and Metabolic Disorders in Skopje, R. Macedonia. We prospectively included 24 consecutive patients (22 female, 2 male) with newly diagnosed SCH, defined by normal FT4 (10.3–24.45 pmol/l)

and elevated TSH (> 4.2 mU/l) levels. Their mean age was 45.37 ± 16.31 years and mean BMI was 27.67 ± 5.69 kg/m².

Thirteen (12 female, 1 male) healthy, euthyroid subjects were included in the study as a control group. Their mean age was 47.85 ± 15.78 years and mean BMI was 27.04 ± 4.46 kg/m². No patients had a previous history of thyroid disease or took any medication related to thyroid disease or lipid metabolism. Patients with diabetes mellitus, liver or renal disease, chronic pancreatitis, primary hyperlipidaemia, ovulatory dysfunction and infertility were excluded from the study. All patients gave informed consent to participate in the study.

Participants had a physical examination and laboratory analyses. Outcome measures were TSH, fT₄, TPOabs, fasting TL, TH, HDL-C, LDL-C, triglycerides, blood pressure, and BMI.

Blood samples were drawn at 08:00am after a 14-hour fast. The blood samples for lipoproteins were analysed using Cobas Integra 700, according to standard methods. TH and triglycerides were determined by full enzymatic methods (TH-CHOD-POD-PAP and triglycerides-GPO; Cobas Integra 700, Hoffmann-La Roche, Basel, Switzerland). HDL-C was measured by the polyanion precipitation method, while LDL-C was calculated using the Friedewald formula. LDL-C were fractionated using ultracentrifugation in cases of triglycerides exceeding 4mmol/l. Serum TSH and free T₄ concentrations were measured using an Immulite 2000 chemiluminescent analyser (Siemens Medical Solutions Diagnostics, Los Angeles, CA, USA). The sensitivity of the assays were 0.004 μ IU/ml and 0.3 ng/dl, respectively. TPOabs was determined by immunometric assay obtained from Diagnostic Products Corporation (Los Angeles, CA). Blood pressure was measured twice in a supine position. In a case of hypertension ($> 145/90$ mmHg) the measurement was repeated after five minutes. The participants were weighed wearing clothes but without shoes in the morning with an electronic scale. Height was measured to the nearest 1 cm with a stadiometer.

Statistical analysis

Statistical analysis was performed using the Statistics for Windows programme, version 5.0. Comparison of the groups was examined using Student's t test. To determine the relationship between TSH and blood pressure, TL, TH, HDL-C, LDL-C, and triglycerides, Spearman's non-parametric correlation test was used. Spearman rank correlation was also used to determine the relationship between blood pressure and lipid profiles in patients with SCH. P values < 0.05 were considered statistically significant.

Results

The results of the thyroid function tests and the personal characteristics of the two groups are shown in table 1. Eight females in the patients group and three females in the control group were in the post-menopausal period. Smoking habits were comparable in the groups. None of these females had hormone replacement therapy at the moment or in the previous couple of years.

Table 1 – Табела 1

Hormonal and personal characteristics of patients with SCH and normal controls
Хормонални и лични карактеристики на пациентите
со СКХ и контролната група

Variables	Subclinical hypothyroidism (n = 24)	Controls (n = 13)	df = 35 t-value	P-value
Sex M : F	2 : 22	1 : 12	0.07	0.94(NS)
Age (years)	45.37 ± 16.31	47.85 ± 15.78	0.45	0.66(NS)
BMI (kg/m ²)	27.67 ± 5.69	27.04 ± 4.46	0.51	0.61(NS)
fT4 pmol/l	14.24 ± 3.38	16,21 ± 2.04	1.91	0.06(NS)
TSH mU/L	8.86 ± 4.28	1,53 ± 0.8	6.13	< 0.0001

Results are mean ± SD; df – degree of freedom; NS – non significant

Резултатите се просек ± СД; ДФ – степени на слобода; НС – нема сигнификантност

The groups were similar with respect to sex, age and BMI (NS in all cases). The patients had a statistically higher TSH levels than the control group ($p < 0.0001$). Positive tests to TPOabs (> 34 iU/ml) were detected in 66.6% of patients and in 7.6% of controls. Patients with SCH had statistically significant higher diastolic blood pressure than the euthyroid, control group (85.24 ± 14.44 vs. 74.17 ± 14.89 mmHg); ($p = 0.04$). Serum mean levels of TL (8.46 ± 2.15 g/l), triglycerides (1.45 ± 0.74 mmol/l), TH (5.32 ± 1.35 mmol/l), HDL -C (1.32 ± 0.29 mmol/l), and LDL -C (3.29 ± 1.2 mmol/l) were not significantly different from the values in the controls (7.78 ± 1.25 g/l, 1.45 ± 0.44 , 5.47 ± 1.01 , 1.33 ± 0.46 , and 3.18 ± 1.02 , respectively) (NS in all cases).

The percentages of patients and controls with elevated blood pressure and abnormal lipid profiles are given in Table 2. The percentage of patients having borderline elevated triglycerides (≥ 2 mmol/l), LDL-C (≥ 3.7 mmol/l), TH/HDL-C (> 5.51) and LDL-C/HDL-C ratios (> 3.46) was significantly higher than in the controls ($p < 0.05$). Hypercholesterolaemia was detected in similar percentages in patients and controls.

Table 2 – Табела 2

Individual percentage analysis of lipid and blood pressure values of patients with SCH and normal controls

Индивидуални процентијални анализи на липидниот профил и крвниот притисок кај СКХ и контролната група

Variables	Subclinical hypothyroidism	Controls
Total lipids ($\geq 10\text{g/l}$)	36.84%	12.5%
Triglycerides ($\geq 2\text{mmol/l}$)	34.78%	7.7%
Total cholesterol ($\geq 5.2\text{mmol/l}$)	47.82%	46.15%
LDL-C ($\geq 3.7\text{mmol/l}$)	41.66%	25%
Total cholesterol/HDL-C (> 5.51)	21.74%	7.7%
LDL-C/HDL-C (> 3.46)	21.7%	0%
A. Hypertension $\geq 140/90\text{mmHg}$	29%	15.3%

A significant positive correlation was observed between blood pressure and TH. Serum triglyceride levels were positively correlated with TH and negatively correlated with HDL-C (Table 3). In patients with SCH, no significant correlations were observed between TSH and TH, TL, triglycerides, LDL-C, HDL-C and blood pressure.

Table 3 – Табела 3

Spearman correlation coefficient between blood pressure and lipid profiles in patients with SCH

Коефициент на Спјерманова ранг корелација помеѓу крвниот притисок и липидниот профил кај испитаниците со СКХ

Parameter	Systolic blood pressure		Diastolic blood pressure		Triglycerides	
	R	p	R	p	R	P
Total cholesterol	0.48	0.01	0.36	0.04	0.57	0.003
LDL-C	0.38	0.06	0.24	0.25	0.35	0.09
HDL-C	0.07	0.75	-0.01	0.97	-0.58	0.003
Triglycerides	0.29	0.16	0.38	0.06	1.00	0.01

Discussion

In the present study we have demonstrated non-significant differences between lipid profiles in patients with SCH and values observed in age-matched

euthyroid controls. However, a significant percentage of these patients had arterial hypertension, hypertriglyceridaemia, elevated LDL-C, elevated TH/HDL-C ratio, and elevated LDL-C/HDL-C ratio as compared with the control group.

Also, we have demonstrated that patients with SCH have significantly higher diastolic blood pressure than the control group. We found that approximately 29% of SCH patients had diastolic hypertension compared with 15.3% in the euthyroid, control group. Luboshitzky *et al.* [9] have shown that women with SCH have a mean diastolic blood pressure higher than control group. This corresponds with our findings. Several studies have reported impaired left ventricular diastolic and systolic myocardial functions in subclinical hypothyroidisms which reverted to normal during L-thyroxine replacement therapy [23–25]. Exposure of aortic endothelial and vascular smooth muscle cells to triiodothyronine (T3) resulted in cellular relaxation. Two binding sites specific for T3 were identified. When cells were exposed to T3, no effect on phosphorylation or nitric oxide production were observed, suggesting that T3 acted directly on the vascular smooth muscle cells to cause vascular relaxation [26]. These data provide our results with a greater physiologic impact and rationale for replacement therapy.

Results of serum lipid concentrations in SCH revealed conflicting data. TH and HDL-C were elevated in several reports, but were not different from those in the controls in most studies [7, 27]. Lower serum HDL-C levels were reported in few studies and were not different from the euthyroid controls in most other studies [7, 28]. Since LDL-C is atherogenic while HDL-C is protective, elevated TH/HDL and LDL/HDL ratios have been used as indexes of increased risk for atherosclerosis. In our study we found that the percentages of patients with atherogenic lipid profiles (TH/HDL-C and LDL-C/HDL-C) were higher than in the controls.

Maas *et al.* [29] have demonstrated the effect of LDL-C on increased atherogenesis in SCH. SCH-related mechanisms, including lipid alteration, have not been exactly established. The cause of these alterations may be increased cholesterol synthesis and decreased activity of hepatic and lipoprotein lipases in thyroid failure [30]. Additionally, decreased cholesterol excretion, a reduced number of LDL receptors on the liver cell surface and decreased plasma LDL receptors are possible mechanisms leading to lipid abnormalities in hypothyroidism [31].

There is a growing body of evidence indicating that elevated triglyceride levels are an independent risk factor for atherosclerosis [32]. Hypertriglyceridaemic patients often develop a lipoprotein profile characterized by elevated triglycerides and LDL-C and low HDL-C [33, 34]. It is estimated that the aggregated risk associated with triglycerides greater than 2.28mmol/L and a TH/HDL-C ratio greater than 5.0 contributes 25% of the cardiovascular events [35].

Although TSH has been suggested as the major factor in the relationship between lipid abnormalities and SCH, we did not observe any correlations between lipid profiles and blood pressure on the one hand and TSH levels in patients with SCH on the other [20]. The only explanation is probably the small sample of participants.

Conclusion

Our study revealed that SCH patients do have a greater prevalence of dyslipidaemia and arterial hypertension, and, as well, a greater value of mean diastolic pressure vs. control patients. This may increase the risk of accelerated atherosclerosis and premature artery disease in some patients that should be investigated in larger longitudinal studies.

REFERENCES

1. Hueston WJ., Pearson WS. (2004): Subclinical Hypothyroidism and the Risk of Hypercholesterolemia. *Ann Fam Med*; 2(4): 351–355.
2. Danese MD., Ladenson PW., Meinert CL., Powe NR. (2000): Effect of Thyroxine Therapy on Serum Lipoproteins in Patients with Mild Thyroid Failure: A Quantitative Review of the Literature. *Clin Endocrinol (Oxf)*; 85(9): 2993–3001.
3. Parle JV., Franklyn JA., Cross KW., Jones SC., Sheppard MC. (1991): Prevalence and follow-up of abnormal thyrotropin (TSH) concentrations in the elderly in the United Kingdom. *Clin Endocrinol (Oxf)*; 34: 77–83.
4. Cappola AR., Ladenson PW. (2003): Hypothyroidism and atherosclerosis. *Journal of Clinical Endocrinology and Metabolism*; 88: 2438–2444.
5. Walsh JP., Bremner AP., Bulsara MK. *et al.* (2005): Thyroid Dysfunction and Serum Lipids: A Community-Based Study. *Clin Endocrinol*; 63(3): 670–675.
6. Miura S., Iitaka M., Yoshimura H. *et al.* (1994): Disturbed lipid metabolism in patients with subclinical hypothyroidism: effect of thyroxine therapy. *Internal Medicine*; 33: 413–417.
7. Kung AWC., Pang RWC., Janus ED. (1995): Elevated serum lipoprotein (a) in subclinical hypothyroidism. *Clinical Endocrinology*; 43: 445–449.
8. Efstathiadou Z., Bitsis S., Milionis HJ. *et al.* (2001): Lipid profile in subclinical hypothyroidism: is L-thyroxine substitution beneficial? *European Journal of Endocrinology*; 145: 705–710.
9. Luboshitzky R., Aviv A., Herer P., Lavie L. (2002): Risk factors for cardiovascular disease in women with subclinical hypothyroidism. *Thyroid*; 12: 421–425.
10. Canaris GJ., Manowitz NR., Major G., Ridgway C. (2000): The Colorado thyroid disease prevalence study. *Ann Intern Med*; 160: 526–534.
11. Jung CH., Sung KC., Shin HSS. *et al.* (2003): Thyroid dysfunction and their relation to cardiovascular risk factors such as lipid profile, hsCRP, and waist hip ratio in Korea. *Korean Journal of Internal Medicine*; 18: 146–153.

12. Parle JV., Franklyn JA., Cross KW., Jones SR., Sheppard MC. (1992): Circulating lipids and minor abnormalities of thyroid function. *Clinical Endocrinology*; 37: 411–414.
13. Pirich C., Mullner M., Sinzinger H. (2000): Prevalence and relevance of thyroid dysfunction in 1922 cholesterol screening participants. *Journal of Clinical Epidemiology*; 53: 623–629.
14. Vierhapper H., Nardi A., Grosser P., Raber W., Gessl A. (2000): Low-density lipoprotein cholesterol in subclinical hypothyroidism. *Thyroid*; 10: 981–984.
15. Kanaya AM., Harris F., Volpato S., Perez-Stable EJ., Harris T., Bauer DC. (2002): Association between thyroid dysfunction and total cholesterol level in an older biracial population. *Archives of Internal Medicine*; 162: 773–779.
16. Langer P., Kocan A., Tajtakova M. *et al.* (2003): Thyroid function and cholesterol level: paradoxical findings in large groups of population with high cholesterol food intake. *Endocrine Regulations*; 37: 175–180.
17. Imaizumi M., Akahoshi M., Ichimaru S. *et al.* (2004): Risk for ischemic heart disease and all-cause mortality in subclinical hypothyroidism. *Journal of Clinical Endocrinology and Metabolism*; 89: 3365–3370.
18. Saito I., Saruta T. (1994): Hypertension in thyroid disorders. *Endocrinol Metab Clin North Am*; 23: 379–386.
19. Streeten DH., Anderson GH., Howland T., Chiany R., Smulyan H. (1998): Effects of thyroid function on blood pressure. Recognition of hypothyroid hypertension. *Hypertension*; 11: 78–83
20. Toruner F., Altinova AE., Karakoc A. *et al.* (2008): Risk factors for cardiovascular disease in patients with subclinical hypothyroidism. *Adv Ther*; 25(5): 430–437.
21. Elder J., St McLelland AJ., O'Reilly D., Packard CJ., Series JJ., Shepherd J. (1990): The relationship between serum cholesterol and serum thyrotropin, thyroxine and tri-iodothyronine concentrations in suspected hypothyroidism. *Annals of Clinical Biochemistry*; 27: 110–113.
22. Staub JJ., Althaus BU., Engler H. *et al.* (1992): Spectrum of subclinical and overt hypothyroidism: effect on thyrotropin, prolactin, and thyroid reserve, and metabolic impact on peripheral target tissues. *American Journal of Medicine*; 92: 631–642.
23. Forfar JC., Wathen CG., Todd WT. *et al.* (1985): Left ventricular performance in subclinical hypothyroidism. *Q J Med*; 22: 857–865.
24. Biondi B., Fazio S., Palmieri EA. *et al.* (1999): Left ventricular diastolic dysfunction in patients with subclinical hypothyroidism. *J Clin Endocrinol Metab*; 84: 2064–2067.
25. Monzani F., DiBello V., Caraccio N. *et al.* (2001): Effect of levothyroxime on cardiac function and structure in subclinical hypothyroidism: A double blind, placebo-controlled study. *J Clin Endocrinol Metab*; 86: 1110–1115.
26. Ojamaa K., Klemperer JD., Klein I. (1996): Acute effects of thyroid hormone on vascular smooth muscle. *Thyroid*; 6: 505–512.
27. Hak AE., Pols HAP., Visser TJ., Drexhage HA., Hofman A., Witteman JCM. (2000): Subclinical hypothyroidism is an independent risk factor for atherosclerosis and myocardial infarction in elderly women: The Rotterdam study. *Ann Intern Med*; 132: 270–278.

28. Althaus BU., Staub JJ., Ryff-de Leche A., Oberhansli A., Stahelin HB. (1988): LDL/HDL-changes in subclinical hypothyroidism: Possible risk factors for coronary artery disease. *Clin Endocrinol*; 28: 157–163.
29. Maas R., Boger RH. (2003): Old and new cardiovascular risk factors: from unresolved issues to new opportunities. *Atheroscler Suppl*; 4: 5–17.
30. Valdemarsson S., Hedner P., Nilsson-Ehle P. (1982): Reversal of decreased hepatic lipase and lipoprotein lipase activities after treatment of hypothyroidism. *Eur J Clin Invest*; 12: 423–428.
31. Duntas LH. (2002): Thyroid disease and lipids. *Thyroid*; 12: 287–293.
32. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP). (2001): Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*; 285: 2486–2497.
33. Brewer HB. (1999): Hypertriglyceridemia: Changes in the plasma lipoproteins associated with an increased risk of cardiovascular disease. *Am J Cardiol*; 83: 3F–12F.
34. Oberman A., Kreisberg RA. (2000): Hypertriglyceridemia and coronary heart disease. *J Clin Endocrinol Metab*; 85: 2098–2105.
35. Assmann G., Schutt H., Von Eckardstein A. (1996): Hypertriglyceridemia and elevated lipoprotein (a) are risk factors for major coronary events in middle aged men. *Am J Cardiol*; 77: 1179–1184.

Резиме

ДИСЛИПИДЕМИЈА И ХИПЕРТЕНЗИЈА КАЈ БОЛНИ СО СУБКЛИНИЧКИ ХИПОТИРОИДИЗАМ

Велкоска Накова В.,¹ Крстевска Б.,¹ Бошевски М.,²
Димитровски Ч.,¹ Серафимоски В.^{3,4}

¹Клиника за ендокринологија и болести на метаболизмот,
Медицински факултет, Скопје, Р. Македонија

²Клиника за кардиоваскуларни заболувања,
Медицински факултет, Скопје, Р. Македонија

³Клиника за тироидно-хипофизна патологија,
Медицински факултет, Скопје, Р. Македонија

⁴Македонска академија на науките и уметностите, Скопје, Р. Македонија

Цел: Да откриеме дали субклиничкиот хипотироидизам е асоциран со дислипидемија и хипертензија.

Материјали и методи: Спроведовме проспективна, контролирана студија на Клиниката за ендокринологија – Скопје, во период од 6 месеци. Во студијата учествуваа 24 консекутивни лица со СКХ и 13 здрави, еутироидни лица, како контролна група. СКХ беше дефиниран како зголемено ниво на тиреостимулирачки хормон (TSH) (> 4,2 mU/l) и нормално ниво на слободен тироксин (fT4) (10,3–

24,45 pmol/l). Ниту еден од учесниците, претходно не беше лекуван со тироксин. На сите учесници им се измери крвен притисок, BMI (индекс на телесна маса), TSH, fT4, антитела против тироидната пероксидаза, вкупни липиди, вкупен холестерол, HDL, LDL холестерол и триглицериди.

Резултати: Просечниот дијастолен крвен притисок беше зголемен кај пациентите со субклинички хипотироизам наспроти контролната група (85 наспроти 74 mmHg; $p < 0,05$). Просечните вредности на вкупните липиди, триглицериди, вкупен холестерол, HDL холестерол и LDL холестерол не се разликуваат кај пациентите со СКХ во споредба со контролната група. Индивидуалните анализи открија дека процентот на пациентите со СКХ кои имаат хипертензија (29%), хипертриглицеридемија (34,78%), зголемен LDL холестерол (41,66%), вкупен холестерол/HDL (21,74%) и LDL/HDL однос (21,7%) се повисоки во однос на истиот процент кај контролната група. Не беше утврдена сигнификантна корелација помеѓу TSH и испитуваните параметри.

Заклучок: Нашата студија откри дека пациентите со СКХ имаат поголема преваленца на дислипидемија и хипертензија, како и повисок просечен дијастолен крвен притисок во споредба со контролната група.

Клучни зборови: субклинички хипотироидизам, дислипидемија, хипертензија, атеросклероза, ризик фактори.

Corresponding Author:

Velkoska Nakova V.
Endocrinology, Diabetes and Metabolic Disorders Clinic,
Faculty of Medicine,
1000 Skopje, R. Macedonia

E-mail: valentina.velkoska@yahoo.com