The Relation between Hypothyroidism and Dyslipidemia in Non-Diabetic Patients

Nabil Abd Elfatah El Kafrawy; Mohamed Abd El Raouf Qorani; Mohamed Zakarya Nouh and Ahmed Shaaban Ahmed Mohammed

Internal Medicine, Faculty of Medicine, Menoufia University
omarahmed92013@yahoo.com

Abstract: **Background**: Hypothyroidismis a common cause of secondary dyslipidemia**.** Hyperlipidemia is known to be present in patients with both primary and secondary hypothyroidism. Several cross-sectional studies have reported that there are increased levels of total cholesterol and low-density lipoprotein (LDL) cholesterol in adults with sub clinical hypothyroidism compared with euthyroid controls. In adults, increasing levels of TSH is associated with increases in total cholesterol, LDL cholesterol and triglycerides and with decreases in HDL cholesterol. The effect of serum thyroid hormones on lipid profile is a complex phenomenon. Thyroid hormone has various effects on both synthesis and degradation of lipids in vivo. Although thyroid hormones decrease serum LDL cholesterol by increasing its clearance through LDL receptors on the liver, low-serum TG is maintained by the stimulation of tissue lipoprotein lipase enzyme. It decreases the serum HDL by increasing the activity of cholesteryl-ester transfer protein (CETP), hepatic lipase, and the expression of HDL receptors on the liver. **The aim of the work:** The aim of the study was to evaluate the relation between hypothyroidism and dyslipidaemia in non-diabetic patients. **Patinets And Methods:** This study was carried out on 50 already diagnosed hypothyroid non diabetic patients regularly coming for management & follow up at Endocrine & diabetic clinic in Menoufia University (group I) and 20 healthy persons as control group (group II). **RESULTS**: Regarding thyroid hormones, there was a significant increase of TSH in patients than the control, while there was a significant decrease of both free T3 and T4 in patients than the control group. There was a positive significant correlation between TSH and serum cholesterol, LDL cholesterol, non HDL cholesterol and LDL/HDL ratio; on the other hand there was a negative significant correlation between free T3 and T4 and serum cholesterol, LDL cholesterol, non HDL cholesterol and LDL/HDL ratio. There was a positive significant correlation between TSH and BMI, while there was a significant negative correlation between free T3 and T4 and BMI.

[Nabil Abd Elfatah El Kafrawy; Mohamed Abd El Raouf Qorani; Mohamed Zakarya Nouh and Ahmed Shaaban Ahmed Mohammed. **The Relation between Hypothyroidism and Dyslipidemia in Non-Diabetic Patients.** *Stem Cell* 2017;8(3):78-85]. ISSN: 1945-4570 (print); ISSN: 1945-4732 (online). http://www.sciencepub.net/stem. 12. doi:10.7537/marsscj080317.12.

Keywords: Relation; Hypothyroidism; Dyslipidemia; Diabetic; Patient

1. Introduction

Hypothyroidism denotes deficient production of thyroid hormone by the thyroid gland and can be primary (abnormality in thyroid gland itself) or secondary/central (as a result of hypothalamic or pituitary disease). It can cause a number of symptoms, such as tiredness, poor ability to tolerate cold, and weight gain. **(Khandelwal D et al, 2012).**

There are two types of hypothyroidism; subclinical hypothyroidism and overt hypothyroidism. Subclinical hypothyroidism (SH) is characterized by mildly elevated serum TSH concentrations, with normal concentrations of serum free triiodothyronine (T3) and thyroxin (T4), without the typical symptoms of thyroid disease. SH prevalence in adults ranges from 4 to 10%. In the pediatric population, the prevalence of this thyroid disorder is estimated to be less than 10 %. **(Aneta Gawlik et al , 2015)**

The burden of SH in India is expected to increase with increasing iodine insufficiency. Studies have shown conflicting results concerning not only the degree of lipid changes in SH but also the effect of thyroxin substitution therapy. The mean total cholesterol and mean LDL levels are significantly increased in SH but there is no significant difference in the mean HDL, VLDL, and triglyceride levels. There is significant reduction in mean T. cholesterol, mean LDL, mean VLDL, and mean triglyceride levels after treatment with thyroxin, while there is no significant difference among the mean HDL levels. **(Ajay Asranna et al, 2012).**

Overt hypothyroidism is defined as a clinical syndrome of hypothyroidism associated with elevated TSH and decreased serum levels of T4 or T3. Serum Total Cholesterol (TC), LDL-C, lipoprotein (a) [Lp (a)], oxi-LDL, Apo B, remnants of VLDL and Chylomicron (CM) levels are increased in overt hypothyroidism, while serum levels of triglyceride, High-Density Lipoprotein Cholesterol (HDL) and VLDL are normal or slightly increased. All of the lipid abnormalities in overt hypothyroidism are reversible with levothyroxine (L-T4) therapy unless the patient

has underlying hyperlipidemia. In overt hypothyroidism, thyroid hormone effects on LDL receptor expression and cholesterol absorption outweigh the effects of decreased hepatic cholesterol synthesis, leading to high serum levels of LDL, Intermediate Density Lipoprotein Cholesterol (IDC), and total cholesterol levels. Additionally, Lipoprotein Lipase (LPL) activity is decreased in hypothyroidism resulted in higher level of VLDL-TG**. (Journal Endocrinal Diabetes, 2014).**

Hypothyroidism has many symptoms and signs including: Coarse and thinning hair, dry skin, brittle nails, a yellowish tint to the skin, slow body movements, cold skin, inability to tolerate cold, feeling tired, sluggish, or weak, memory problems, depression, or problems concentrating, constipation, heavy or irregular menstrual periods that may last longer than 5 to 7 days. **(Web MD Medical Reference from Healthwise,2014)** The diagnosis of hypothyroidism when suspected can be confirmed with blood tests measuring thyroid stimulating hormone (TSH), free T4 and free T3.

Hypothyroidism can be treated with manufactured levothyroxine; the dose is adjusted according to TSH levels.

Dyslipidemia is a condition that occurs when lipid levels in the bloodstream are too high or low. **(Gordon H et al, 2013)**. It is more common in hypothyroidism. Dyslipidemia is the most important risk factor for atherosclerosis. Atherosclerosis can lead to serious problems, including heart attack, stroke, or even death. (**US National Institutes of Health 2014).**

Dyslipidemia is a common metabolic abnormality in patients with thyroid disease, either in the overt or subclinical forms of the disease, and constitutes the end result of the effect of thyroid hormones in all aspects of lipid metabolism leading to various quantitative and/or qualitative changes of triglycerides, phospholipids, cholesterol, and other lipoproteins. In thyroid disease, dyslipidemia and the coexisting metabolic abnormalities, in combination with the thyroid hormone-induced hemodynamic alterations, explain the high risk for cardiovascular disease. **(Angeliki Chroni, 2011).**

Levothyroxine replacement therapy was administered and patients were assessed every 3 to 4 months for an effect on lipid profile and body mass index during the study period. In both subclinical and overt hypothyroidism associated with dyslipidemia, replacement therapy with levothyroxine resulted in reversal to normal. **(Amit Saxena et al, 2013).**

The aim of the study was to evaluate the relation between hypothyroidism and dyslipidemia in non-diabetic patients.

2. Subjects and methods

Subjects:

This study was carried out at the Endocrinology Unit, Menoufyia University Hospital - Internal Medicine Department on 50 patients with Hypothyroidism already diagnosed and 20 healthy volunteers as controls, between Novembers 2015 and December 2016.

The study was conducted on two groups:-

- (Group A) Fifty Hypothyroid patients.
- (Group B) Twenty healthy adults were used as a control group.

Informed consent from all patients and controls was obtained in accordance with the local ethical committee.

Methods:

All the patients and controls were subjected to the following:

❖ **Full history taking including:**

1. Personal data including age and sex.
2. Age of onset.
3. Family history of hypothyroidism.
4. Duration of hypothyroidism.
5. Thyroid replacement therapy (L-Thyroxin).
6. History of cardiovascular complications.
7. Medications affecting lipid metabolism.
8. History of other medical diseases.

Clinical examination was thoroughly performed to all the patients and controls including:

General examination:

1. General appearance.
2. Body built.
3. Vital signs.

i. Blood pressure: there is a variation in blood pressure measurement.

ii. Pulse: weak pules with decrease force and volume.

iii. Temperature: almost always lower than normal.

iv. Respiratory rate and rythm.

4. Head and neck: for thyroid enlargement and enlarged lymph nodes.
5. Skin and extremities.
6. Heart examination.
7. **N**ervous system.
8. Chest examination.
9. Abdominal examination.

❖ **Laboratory investigations including**

- Consent was taken for each subject and they were requested to fast overnight.
- Blood samples were collected by venepuncture from patients.
- The samples were allowed to clot and the Serum separated by centrifugation at 10,000 rpm for 15 minutes at room temperature.

- Serum samples were stored at −20˚C until tested.
- Blood samples were treated as follows:
- Serum fasting blood glucose and post prandial was done using Spectrophotometer (Spectra scan UV 2600, chemito).
- For Free T3, T4, TSH and lipid estimation 4 ml of blood was taken in a plain test tube and was separated by centrifugation.
- Serum lipids (triglyceride, total cholesterol and HDL cholesterol) were measured in the BIRDEM Biochemistry laboratory, by enzymatic method and mentioned briefly.
- The LDL cholesterol was calculated from observed triglyceride, total cholesterol and HDL cholesterol by using Friedwald's formula (If triglyceride result is below 400 mg/dL).
- If triglyceride result is upon 400 mg/dL used direct LDL-C measurement method follow. The normal value of serum FT4 = (11.0 - 24.0) pmol/L and TSH = (0.40 - 5.0) IU/mL. Desirable levels of blood fats are: total cholesterol; below 200 mg/dL, HDL cholesterol; Men: above 40 mg/dL, Women: above 50 mg/dL. LDL cholesterol below 100 mg/dL.
- CBC.
- Urea and Creatinine.
- Liver enzymes.

3. Results

This study was carried out on 50 hypothyroid already diagnosed non diabetic patients regularly coming for management & follow up at Endocrine & diabetic clinic in Menoufia University (Group I) and 20 healthy persons as control group (Group II).

The results obtained were collected tabulated and analyzed statistically to fulfill the aim of the work as follows:

Table (1), shows the comparison between the two studied groups regarding demographic data. The age in group I (patients group) was 44.62±4.57 years, while in control group was 41.8±6.86 years, by comparing the two groups it was found that there was no statistically significant difference between the two groups regarding age. The age of onset in patients was ranged from 27-48 years with a mean of 38.9±5.18 years, the disease duration was ranged from 1-10 years with a mean of 6.22±1.81, in patients group the positive family history was found in 19 patients (38.0%).

The body mass index in patients was 28.59±4.35, while in control was 22.41±2.86, there was a statistically significant increase in BMI in patients than the control group ($p < 0.05$).

Table (2), show the comparison between the two studied groups regarding lipid profile, the serum cholesterol in patients was 246.52±30.54, while in control was 191.6±30.85, there was a statistically significant increase in serum cholesterol in patients than the control ($p < 0.05$). Serum triglyceride inpatients was 194.38±81.93, while in control was 188.20±80.44, there was an increasing in serum triglyceride in patients but this increasing was statistically insignificant ($p > 0.05$). HDL cholesterol in patients was 40.55±6.29 and in control was 45.40±8.11, there was a statistically significant increase in HDL in control than the patients ($p < 0.05$). LDL cholesterol in patients was 198.22±51.78, while in control was 139.65±31.68, there was statistically significant increase in LDL in patients than the control group ($p < 0.05$). Non HDL cholesterol in patients was 153.17±29.24, and in control was 145.75±31.00, there was no significant difference between the two groups regarding non HDL cholesterol, finally LDL/HDL ratio in patients was 5.18±1.55, while in control was 3.16±0.98, and there was a statistically significant increase in LDL/HDL ratio in patients than the control ($p<0.05$).

Table (3), show the comparison between the two studied groups regarding thyroid hormones. It was found that there was a statistically significant increase of TSH in patients than the control, while there was a significant decrease of both free T3 and T4 in patients than the control group ($p < 0.05$).

Table (4), show the Comparison between the two studied groups regarding blood glucose and post prandial glucose level, there was no statistically significant difference between the two groups regarding fasting blood glucose and 2 hour post prandial blood level ($p > 0.05$).

Table (5), show the comparison between the two studied groups regarding blood picture, it was found that there was no significant difference between the two studied groups regarding blood picture ($p > 0.05$).

Table (6), show the comparison between the two studied groups regarding liver and kidney function, it was found that there was no significant difference between the two studied groups regarding kidney and liver function.

Table (7), show the correlation between thyroid hormones and lipid profile, it was found that there was a positive significant correlation between TSH and serum cholesterol, LDL cholesterol, non HDL cholesterol and LDL/HDL ratio, on the other hand there was a negative significant correlation between free T3 and T4 and serum cholesterol, LDL cholesterol, non HDL cholesterol and LDL/HDL ratio ($p < 0.05$).

Table (1): Comparison between the two studied groups regarding demographic data.

	Group I "patients"	**Group II "control"**	**P**
Age (years) Range Mean S.D.	 35-50 44.62 4.57	 33-52 41.80 6.86	 0.062
Sex **Male** **Female**	 15 (30.0%) 35 (70.0%)	 7 (35.0%) 13 (65.0%)	 0.411
Age of onset (years) Range Mean S.D.	 27-48 38.90 5.18	 -	 -
Duration of disease (years) Range Mean S.D.	 1-10 6.22 1.81	 -	 -
Positive Family history	19 (38.0%)	-	-
BMI (Kg/m^2) Range Mean S.D.	 19.12-34.51 28.59 4.35	 18.5-27.23 22.41 2.86	 0.011*

Table (2): Comparison between the two studied groups regarding lipid profile.

	Group I "patients"	**Group II "control"**	**P**
Serum cholesterol (mg/dl) Range Mean S.D.	 195-318 246.52 30.54	 140-240 191.60 30.85	0.001*
Serum triglyceride (mg/dl) Range Mean S.D.	 103 – 460 194.38 81.93	 90-350 188.20 80.44	0.120
HDL cholesterol (mg/dl) Range Mean S.D.	 30 – 56 40.55 6.29	 35-60 45.40 8.11	0.012*
LDL cholesterol (mg/dl) Range Mean S.D.	 125-320 198.22 51.78	 90-170 139.65 31.68	0.001*
Non HDL Cho. (mg/dl) Range Mean S.D.	 104 – 224 153.17 29.24	 95-185 145.75 31.00	0.212
LDL/HDL (mg/dl) Range Mean S.D.	 2.46-8.16 5.18 1.55	 1.6-4.8 3.16 0.98	0.001*

Table (3): Comparison between the two studied groups regarding thyroid hormones.

	Group I "patients"	**Group II "control"**	**P**
TSH (0.4 – 4.2 mlu/L) Range Mean S.D.	 7.857-81.3 24.68 16.81	 0.31-4.5 1.51 1.36	0.0001*
Free T3(3.5-6.5 pmol/L) Range Mean S.D.	 0.1-2.9 1.54 0.74	 2.3-4.5 3.25 0.67	0.001*
Free T4 (11-24 pmol/L) Range Mean S.D.	 0.13-2.83 0.79 0.53	 0.81-1.7 1.04 0.28	0.005*

Table (4): Comparison between the two studied groups regarding blood glucose and post prandial glucose level.

	Group I "patients"	**Group II "control"**	**P**
FBG (mg/dl) Range Mean S.D.	 70-110 86.34 11.72	 70-115 92.95 13.79	0.088
2hPP (mg/dl) Range Mean S.D.	 70-130 99.02 16.71	 85-140 107.40 18.32	0.095

Table (5): Comparison between the two studied groups regarding blood picture

	Group I "patients"	**Group II "control"**	**P**
Hb (g/dl) Range Mean S.D.	 11.5-16.7 13.67 1.35	 11-18 13.67 2.14	0.498
WBCs (10^3)/ml Range Mean S.D.	 4-11 7.32 2.43	 4-10 7.35 1.87	0.478
Platlets (10^3)/ml Range Mean S.D.	 150-400 275.60 71.36	 150-400 265.55 82.19	0.318

Table (6): Comparison between the two studied groups regarding liver and kidney function

	Group I "patients"	Group II "control"	P
SGOT (u/L) Range Mean S.D.	 10-40 20.58 10.71	 5-40 22.35 11.20	0.275
SGPT (u/L) Range Mean S.D.	 13-40 25.16 8.91	 10-40 25.80 11.08	0.410
Urea (mg/dl) Range Mean S.D.	 10-40 27.74 8.23	 20-40 30.20 7.21	0.112
Creatinine (mg/dl) Range Mean S.D.	 0.5-1.4 0.98 0.23	 0.7-1.25 0.93 0.15	0.155

Table (7): Correlation between thyroid hormones and lipid profile.

		TSH	Free T3	Free T4
serum cholesterol	Pearson Correlation	.885**	-.972**	-.946**
	P-value	.000	.000	.000
serum triglyceride	Pearson Correlation	.198	-.079	-.169
	P-value	.168	.586	.242
HDL cholesterol	Pearson Correlation	.244-	-.147-	-.197-
	P-value	.087	.310	.170
LDL cholesterol	Pearson Correlation	.365**	-.421**	-.395**
	P-value	.009	.002	.005
Non HDL Cho.	Pearson Correlation	.460**	-.217	-.382**
	P-value	.001	.130	.006
LDL/HDL	Pearson Correlation	.469**	-.438**	-.452**
	P-value	.001	.001	.001

4. Discussion

In our study, the mean age in group I (patients group) was 44.62±4.57 years, while in control group was 41.8±6.86 years, on comparing the two groups it was found that there was no statistically significant difference between the two groups regarding age. The body mass index in patients was 28.59±4.35, while in control was 22.41±2.86, there was a statistically significant increase in BMI in patients than the control group.

In agreement with our study **the Pradeep et al. 2013** study was found that the mean age of patients in control, subclinical & overt hypothyroid groups are around 40 years, which is the age of seeking medical advice. These patients are more prone to cardiovascular complications and other problems, which can be delayed by early lifestyle interventions and treatment. In our study the majority of patients are females. In agreement with our study are the studiesof **Agarwal et al, 2011** that have found that the hypothyroidism is more common in women than men.

In our study, there was no statistically significant difference between the two groups regarding fasting blood glucose; two hours post prandial blood level, blood picture, kidney and liver function.

In agreement in our study **Dr. Sapna Goyal et al, 2013** study has found that there was no significant difference between control, subclinical hypothyroidism and overt hypothyroidism as regarding fasting blood glucose.

In disagreement in our study the **Singh et al. 2010** studies have found that the mean fasting blood sugar is slightly higher in Subclinical & Overt hypothyroid groups than the control group. This is in consistence with our study findings.

In our study, the serum cholesterol in patients was 246.52±30.54, while in control was 191.6±30.85, there was a statistically significant increase in serum cholesterol in patients than the control.

In agreement with our study **the Goswami B, Bo Abbas Y and Bahceci M** studies have found that there is a significant increase in serum cholesterol in

subclinical hypothyroidism and overt hypothyroidism than in control.

In our study Serum triglyceride inpatients was 194.38±81.93, while in control was 188.20±80.44, there was an increasing in serum triglyceride in patients but this increasing was insignificant.

In agreement with our study as regarding triglyceride **Al Sayed A et al. 2006 and Tuzcu et al. 2005** have found that there was an increasing in serum triglyceride in subclinical and overt hypothyroidism patients than control but this increasing was insignificant.

In disagreement with our study as regarding triglyceride **Singh et al. 2010** have found that there is a significant increasing in the triglyceride in subclinical and overt hypothyroid patients than in the control and the level of Triglyceride is rising with age in all the groups. This suggests that increasing grades of hypothyroidism and increasing age leads to increase in Triglyceride levels.

In our study, HDL cholesterol in patients was 40.55±6.29 and in control was 45.40±8.11; there was a statistically significant increase in HDL in control than the patients. LDL cholesterol in patients was 198.22±51.78, while in control was 139.65±31.68, there was statistically significant increase in LDL in patients than the control group. Non HDL cholesterol in patients was 153.17±29.24, and in control was 145.75±31.00, there was no significant difference between the two groups regarding non HDL cholesterol, LDL/HDL ratio in patients was 5.18±1.55, while in control was 3.16±0.98, and there was a statistically significant increase in LDL/HDL ratio in patients than the control.

In agreement with our study **the Pradeep et al. 2013** study has found that there is increase in the mean LDL Cholesterol in subclinical and overt hypothyroid patients than the normal control.

As regarding HDL **Singh et al 2010** study has found that the HDL was significantly increasing in the control than in the subclinical and overt hypothyroid patients and this consistence with our study finding. This demonstrates that with increasing grades of hypothyroidism there is decrease in serum HDL values. The level of HDL Cholesterol is decreasing with age in all the groups.

In disagreement with our study as regarding HDL **Tuzcu et al 2005** and **Al Sayed A et al 2006** have found that there is an increasing in HDL in the subclinical and overt hypothyroid patients than the control and this increasing was insignificant.

In our study, regarding thyroid hormones, there was a significant increase of TSH in patients than the control, while there was a significant decrease of both free T3 and T4 in patients than the control group. There was a positive significant correlation between TSH and serum cholesterol, LDL cholesterol, non HDL cholesterol and LDL/HDL ratio; on the other hand there was a negative significant correlation between free T3 and T4 and serum cholesterol, LDL cholesterol, non HDL cholesterol and LDL/HDL ratio. There was a positive significant correlation between TSH and BMI, while there was a significant negative correlation between free T3 and T4 and BMI. There was a positive significant correlation between 2 hour PP and TSH, and there was a negative significant correlation between free T3 and T4 and 2 hour PP. There was no significant correlation between thyroid hormones and liver and kidney function.

The correlation of TSH values with Serum Cholesterol, Serum Triglyceride, Serum LDL Cholesterol, in the normal control population is statistically not significant but in subclinical hypothyroidism and overt hypothyroidism these are statistically significant.

In agreement with our study **the Pradeep et al. 2013** study the correlation of TSH values with Serum HDL Cholesterol in the normal control population & subclinical hypothyroidism is statistically not significant. In overt hypothyroidism it is statistically significant.

In disagreement with our study **Lu et al. 2011** has found that in euthyroid population there were no significant correlations between TSH and serum Total Cholesterol, Triglyceride, HDL-C and LDLC.

Most of the studies there is similar findings to our study suggesting positive correlation between TSH and T Cholesterol, LDL-C and Triglyceride. There is negative correlation between TSH and HDL-C. This finding further suggests that increasing grades of hypothyroidism causes dyslipidemia.

In hypothyroidism there is decrease in energy metabolism and heat production. It is reflected by low basal metabolic rate, decreased appetite, cold intolerance, and slightly low basal body temperature. **(Kronenberg HM et al, 2008).**

In hypothyroidism biosynthesis of fatty acids and lipolysis are reduced. The lipid changes bear in general a reciprocal relationship to the level of thyroid activity. The increased serum cholesterol may represent an alteration in a substrate steady state level caused by a transient proportionally greater retardation in degradation than in synthesis.

The increase of serum cholesterol is largely accounted for by an increase of LDL-cholesterol, which is cleared less efficiently from the circulation due to a decreased T3-dependent gene expressing of the hepatic LDL-receptor.

Interestingly, the LDL particles of hypothyroid patients are also susceptible to increased oxidizability. The changes in plasma LDL and HDL-cholesterol are related to changes in free thyroxine, not to

polymorphisms in LDL receptor or cholesteryl ester transfer protein genes. The increase of serum triglycerides has been related to a decreased lipoprotein lipase activity. Lipoprotein (a) is increased in hypothyroidism in some but not all studies. Remnant particles in serum (reflecting chylomicron and VLDL remnants) are less effectively cleared in hypothyroidism. Thus, the changes in plasma lipids in hypothyroidism result in an atherogenic lipid profile.

Conclusions

The study has concluded that, Hypothyroidism causes dyslipidemia. Thus, it may be a good practice to screen patients with hypothyroidism for evidence of metabolic syndrome and in preventing various other complications. The screening and treatment for subclinical hypothyroidism should be done to prevent its adverse effects on lipid metabolism. Thyroid hormones regulate the expression of enzymes involved in all steps of lipid metabolism leading to the development of qualitative and quantitative changes of lipids, in thyroid disease.

References

1. Aneta Gawlik, Kamila Such, Aleksandra Dejner, Agnieszka Zachurzok, Aleksandra Antosz, and Ewa Malecka-Tendera. International Journal of Endocrinology, subclinical hypothyroidism in children and adolescents. 2015, article ID 691071, 12 pages.
2. Ajay Asranna,RS Taneja,Bindu Kulshreshta. Dyslipidemia in subclinical hypothyroidism and the effect of thyroxine on lipid profile.2012 volume: 16 Issue: 8 Page: 347-349.
3. Journal ndocrinal Diabetes & obesity. Update on Lipid Metabolism and Thyroid Disorders. 19 July 2014 ISSN: 2333-6692.
4. Web MD Medical Reference from Healthwise, signs and Symptoms of hypothyroidism 2014.
5. Gordon H. Williams, MD, William F. Young, Jr. Endocrine causes of dyslipidemia 2013.
6. Koba S, Hirano T, Nihon Rinsho. Dyslipidemia and atherosclerosis. pupmed 2011 Jan; 69(1):138-43.
7. Amit Saxena, Pragati Kapoor, Shikha Saxena and A K Kapoor. Internet Journal of medical update, effect of levothyroxine therapy on dyslipidemia in hypothyroid patients. 2013 July;8(2):39-49.
8. Khandelwal D, Tandon N; Overt and subclinical hypothyroidism: who to treat and how. Drugs. 2012 Jan 1 72(1):17-33.
9. Pradeep Sharma1, Dibyaratna Patgiri, Dr. Sapna Goyal, Dr. Geeta Sharma, M. S. Pathak. Hypothyroidism causing dyslipidemia in both subclinical & overt Hypothyroidism. Indian Journal of Basic & Applied Medical Research; June 2013: Issue-7, Vol.-2, P. 779-788.
10. Agarwal G, Sudhakar MK, Singh M, Senthil N, Rajendran A. The prevalence of thyroid dysfunction among South Indian women with Metabolic Syndrome. J. of clinical & diagnostic research 2011; 5(2): 213-216.
11. Singh BM, Goswami B and Mallika V. Association between insulin resistance and hypothyroidism in females attending a tertiary care hospital. Indian Journal of Clinical Biochemistry 2010; 25 (2): 141-145.
12. Al Sayed A, Al Ali N, Bo Abbas Y, Alfadhli E. Subclinical hypothyroidism is associated with early insulin resistance in Kuwaiti women. Endocrine Journal 2006; 53: 653-657.
13. Kronenberg HM, Melmed S, Polonsky KS, Larsen PR (editors). Williams Textbook of Endocrinology. Chapter: Hypothyroidism and thyroiditis. 11ed. Elsevier 2008; 337-405.

5/31/2017