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Correlation between Serum Lipid Profile & Carotid Intima Media Thickness among Hypertensive Patients in a Tertiary Care Institution – A hospital Based Study

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ABSTRACT

Background: In this study, we wanted to evaluate the association between hypertension and carotid intima media thickness (CIMT), determine the relationship of serum lipid profile with carotid intima media thickness in hypertensive patients, and assess the association between hypertension and dyslipidaemia.

Materials and methods: This was a hospital based cross-sectional observational study conducted among 100 patients who presented with hypertension to the Department of Medicine at FAAMCH, Barpeta, Assam, from September 2020 to August 2021 after obtaining clearance from Institutional Ethics Committee and written informed consent from the study participants.

Results: The patients were mostly in the high age groups (≥60 years) (47.0%). The body mass index (BMI) of patients ranged from 19.6-33.2 kg/m^2 with mean (± SD) 24.64 ± 2.51 kg/m^2 and median 24.0 kg/m^2. The serum triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDLC) and low-density lipoprotein cholesterol (LDLC) of patients ranged from 51-349, 93-256, 10-82 and 16-196 mg/dl, respectively with mean (± SD) 137.78 ± 49.27, 155.03 ± 41.55, 46.86 ± 17.43 and 82.37 ± 37.85 mg/dl, and median 129, 149, 45 and 73 mg/dl. The LDLC/HDLC ratio of patients ranged from 0.3-8.9 with mean (± SD) 2.08 ± 1.30 and median 2.0.CIMT classification (normal: ≤ 0.90 and abnormal: >0.90 mm), 69 (69.0%) patients were normal and 31 (31.0%) were abnormal. Lipid profile viz. TG (r=0.26, $P < 0.05$), TC (r=0.22, $P < 0.05$), LDL-C (r=0.29, $P < 0.01$) and LDL-C/HDL-C (r=0.30, $P < 0.01$) showed significant. Significant and positive correlation was found between systolic blood pressure (SBP) and diastolic blood pressure (DBP) (r=0.66, $P < 0.001$), and SBP and isolated systolic HTN (r=0.57, $P < 0.001$). Further, DBP also showed significant and positive correlation with LDL-C (r=0.21, $P < 0.05$).

Conclusion: Hypertension and dyslipidaemia are important risk factors for increasing CIMT in high risk group. Multiple regression analysis shows that among the multiple lipid profiles, the most important factor for increasing CIMT is LDL: HDL. Isolated systolic hypertension presented with significantly higher CIMT values. Therefore, patients with isolated systolic hypertension should alert physicians to perform a more thorough evaluation of the cardiovascular profile of such patients. We conclude that the results support the importance of screening hypertensive patients by carotid ultrasound Doppler to assess the ongoing atherosclerotic changes in vessel wall.

Keywords: Serum Lipid Profile, Carotid Intima Media Thickness, Hypertensive Patients.

INTRODUCTION

Hypertension and dyslipidaemia are key risk factors for cardiovascular disease (CVD), and the global prevalence of hypertension is expected to rise, particularly in emerging nations. In recent years, rapid urbanization, increased life expectancy, unhealthy diet, and lifestyle changes have led to an increased rate of CVD in Southeast Asia, including India.[1] Epidemiological studies have established a strong association between hypertension and coronary artery disease.[2] According to the World Health Organization, 1.13 billion people suffer with hypertension globally, with the majority (two-thirds) living in low- and middle- income nations. In 2015, one in every four men and one in every five women had hypertension. Only about 1 in every 5 patients with hypertension has their condition under control. Hypertension is a leading cause of death in the world. One of the global targets for non-communicable diseases is to reduce the prevalence of hypertension by 25% by 2025 (baseline 2010).[3]Between 1980 and 2008, global mean total cholesterol changed minimally,

declining by less than 0.1 mmol/L every decade in both men and women.[4]The WHO region of Europe has the highest prevalence of increased total cholesterol (54 percent for both sexes), followed by the WHO region of the Americas (48 percent for both sexes). The lowest percentages (22.6 percent for AFR and 29.0 percent for SEAR) were found in the WHO African and South East Asian Regions.[4] The prevalence of dyslipidaemia in these studies ranges from 10 – 15% in rural communities to 25 – 30% in urban populations.[5]Austin and colleagues described atherogenic dyslipidaemia (AD) as a clinical condition characterized by elevated serum triglyceride (TG) levels and small-dense low-density lipoprotein (SD LDL) particles with low levels of high-density lipoprotein cholesterol (HDL-C) in 1990, highlighting the atherogenic lipoprotein phenotype.[6] Furthermore, increased levels of large TG-rich vary low-density lipoproteins (VLDL) and apolipoprotein B (Apo B) and decreased levels of small high-density lipoproteins play a key role in atherogenic dyslipidaemia (AD).[7] Atherosclerosis is considered a primary cause of cardiovascular morbidity and mortality. Atherosclerosis is a slowly progressive disease with multiple modifiable and non-modifiable risk factors. Common modifiable risk factors include diabetes mellitus, hypertension, smoking and dyslipidaemia. Dyslipidaemia is the key risk factor fordevelopment of atherosclerosis and therefore contributes significantly to the development of cardiovascular disease (CVD).[8]The rise in serum levels of pro-atherogenic lipoproteins, particularly low density lipoprotein cholesterol (LDL-C) and triglycerides, is one of the fundamental causes of atherosclerosis. A decline in the anti-atherogenic lipoprotein high density lipoprotein (HDL-C) raises the atherosclerotic risk in addition to an increase in LDL-C as an aetiology in atherosclerosis.[9] Castelli Risk Index (CRI-I) and (CRI-II) calculated as (TC/HDL-C) and (LDL- C/HDL-C) respectively, are fractions that have been known to be independent risk factors for atherosclerosis.[10] Atherosclerosis is a systemic disease that mostly affects elastic arteries (carotid, aorta, and iliac arteries) as well as big and medium-sized muscle arteries.

Hypertension is a risk factor for the development of atherosclerosis. In patients with hypertension, alterations to the arterial wall indicate an early involvement of the target organs. [11]Complications of hypertension are either due to sustained elevation of blood pressure (with consequent changes in the vasculature and heart) or due to atherosclerosis that accompanies it. Hypertension and atherosclerosis are two distinguished entities, but hypertension plays an important role in the pathogenesis of atherosclerosis. [11] The carotid intima-media thickness (CIMT) is a well-established technique for detecting and assessing atherosclerosis development. CIMT rises when atherosclerosis spreads throughout the body, allowing for inferences about additional unstudied and possibly difficult-to-reach blood channels, such as the coronary arteries.[12]A meta-analysis of eight major population studies including 37,000 individuals found that a 0.1mm difference in C-IMT is associated with a 10% to 15% increased risk of myocardial infarction and stroke (13-18 percent).[13]Ultrasound measurements of IMT and plaque occurrence in the carotid arteries are important as the extent of arteriosclerosis in these vessels reflects the severity of arterial damage in other vascular territory.[14]High resolution ultrasonography is a non-invasive, simple, safe (non-ionizing) inexpensive, precise, and reproducible method of examining and evaluating the walls of carotid arteries. Extra cranial carotid arteries are chosen for IMT assessment because of their superficial location, easy accessibility, adequate size, and limited movement.[14]Considering the fact that the incidence and prevalence of hypertension is increasing in the northeast population of India and that most of the studies on hypertension with dyslipidaemia and CIMT are based on foreign population with lifestyle different from that of the North East Indian population, the present study will focus on the serum lipid profile and its association with CIMT in hypertensive population of lower Assam which may help prognosticate the progression of hypertension to CAD and CVA.

AIMS AND OBJECTIVES

a) To determine the relationship of serum lipid profile with carotid intima media thickness in hypertensive patients.
b) To assess the association between hypertension and dyslipidaemia.

MATERIALS AND METHODS

This was a hospital based cross-sectional observational study conducted among 100 patients who presented with hypertension to the Department of Medicine at FAAMCH, Barpeta, Assam, from September 2020 to August 2021 after obtaining clearance from Institutional Ethics Committee and written informed consent from the study participants.

INCLUSION CRITERIA

All hypertensive patients attending the general medicine out-patients/admitted to General medicine ward in Fakhruddin Ali Ahmed Medical College and Hospital.

EXCLUSION CRITERIA

- Age less than 18 years.
- Patients on antihypertensive medications.

- K/C/O diabetes mellitus, hypothyroidism.
- H/O intake of any lipid lowering drugs.

STATISTICAL METHODS

Data were summarized as mean ± SD (standard deviation). Groups were compared by one-way analysis of variance (ANOVA) and the significance of mean difference between(inter) the groups was done by Tukey's HSD (honestly significant difference) post hoc test after ascertaining normality by Shapiro-Wilk's test and homogeneity of variance between groups by Levine's test. Pearson correlation analysis was done to assess the association between the variables. A two-tailed (α=2) $P < 0.05$ was considered statistically significant. Analysis was performed on Statistical Package for Social Sciences (SPSS) software (Windows version 22.0).

RESULTS

	Variable	**Total Patients (n=100) (%)**
Age Distribution (Age in Years)	30-40	7 (7.0)
	40-50	17 (17.0)
	50-60	29 (29.0)
	≥60	47 (47.0)
	Variable	**Total Patients (n=100) (%)**
Sex Distribution	Female	50 (50.0)
	Male	50 (50.0)
Table 1: Demographic Distribution		

The distribution of age of patients was summarized in number (n) and percentage (%). The age of patients ranged from 30-85 years with mean (± SD) 57.35 ± 11.68 years and median 57 years. The patients were mostly high age groups (≥60 years) (47.0%). The distribution of gender of patients was summarized in number (n) and percentage among patients, 50.0% were females and 50.0% were males.

The BMI of patients ranged from 19.6-33.2 kg/m^2 with mean (± SD) 24.64 ± 2.51 kg/m^2 and median 24.0 kg/m^2. According to BMI classification, 29 (29.0%) patients were normal weight, 63 (63.0%) over weight, 6 (6.0%) obesity I and 2 (2.0%) obesity II.

	Variable	**Total Patients (n=100) (%)**
Distribution of BMI of Study Population	BMI (kg/m^2):	
	Normal weight (18.5-22.9)	29 (29.0)
	Overweight (23.0-27.9)	63 (63.0)
	Obesity I (28.0-32.9)	6 (6.0)
	Obesity II (33.0-37.9)	2 (2.0)
	Variable	**Total Patients (n=100) (%)**
Distribution of SBP of Study Population	SBP (mmHg):	
	Stage 1 hypertension (140-159)	68 (68.0)
	Stage 2 hypertension (≥160)	32 (32.0)
	Variable	**Total Patients (n=100) (%)**
Distribution of DBP of Study Population	DBP (mmHg):	
	Pre-hypertension (80-89)	9 (9.0)
	Stage 1 hypertension (90-99)	66 (66.0)
	Stage 2 hypertension (≥100)	25 (25.0)
	Variable	**Total Patients (n=100) (%)**
Distribution of HTN of Study Population	HTN:	
	Stage I HTN	65 (65.0)
	Stage II HTN	26 (26.0)
	Isolated Systolic HTN	9 (9.0)
Table 2		

The SBP and DBP of patients ranged from 140-210 and 80-120 mmHg respectively with mean (± SD) 159.32 ± 13.53 and 94.94 ± 5.99 mmHg respectively and median 156 and 94 mmHg respectively. According to SBP, 68 (68.0%) patients were stage 1 hypertension and 32 (32.0%) were stage 2 hypertensions. However, according to DBP, 9 (9.0%) patients were pre hypertension, 66 (66.0%) stage 1 hypertension and 25 (25.0%) were stage 2 hypertensions.

Moreover, according to JNC-8 classification of blood pressure (SBP and DBP) for hypertension (HTN), 65 (65.0%) patients were stage I HTN, 26 (26.0%) stage II HTN and 9 (9.0%) isolated systolic HTN.

	Variable	**Total Patients (n=100) (%)**
Distribution of TG of Study Population	TG (mg/dl):	
	Normal (<150)	71 (71.0)
	Borderline high (150-199)	19 (19.0)
	High (200-499)	10 (10.0)
	Variable	**Total Patients (n=100) (%)**
Distribution of TC of Study Population	TC (mg/dl):	
	Normal (<200)	82 (82.0)
	Borderline high (200-239)	15 (15.0)
	High (≥240)	3 (3.0)
	Variable	**Total Patients (n=100) (%)**
Distribution of HDLC of Study Population	HDLC (mg/dl):	
	Low (<40)	33 (33.0)
	Optimal (40-60)	37 (37.0)
	High (≥60)	30 (30.0)
Table 3		

The TG, TC, HDLC and LDLC of patients ranged from 51-349, 93-256, 10-82 and 16-196 mg/dl respectively with mean (± SD) 137.78 ± 49.27, 155.03 ± 41.55, 46.86 ± 17.43 and 82.37 ± 37.85 mg/dl respectively and median 129, 149, 45 and 73 mg/dl respectively.

According to standard classification of TG, 71 (71.0%) patients were normal, 19 (19.0%) borderline high and 10 (10.0%) high. According to TC, 82 (82.0%) patients were normal, 15 (15.0%) borderline high and 3 (3.0%) high. Similarly, HDLC show 33 (33.0%) patients were with low HDLC, 37 (37.0%) optimal and 30 (30.0%) high.

LDL-C showed 73 (73.0%) patients were with optimal LDL-C, 11 (11.0%) above optimal, 12 (12.0%) borderline high, 3 (3.0%) high and 1 (1.0%) very high. The CIMT values of patients ranged from 0.54-1.30 mm with mean (± SD) 0.84 ± 0.14 mm and median 0.84 mm. According to CIMT classification (normal: ≤ 0.90 and abnormal: >0.90 mm), 69 (69.0%) patients were normal and 31 (31.0%) were abnormal.

	Variable	**Total Patients (n=100) (%)**
Distribution of LDLC of study population	LDLC (mg/dl):	
	Optimal (<100)	73 (73.0)
	Above optimal (100-129)	11 (11.0)
	Borderline high (130-159)	12 (12.0)
	High (160-189)	3 (3.0)
	Very high (≥190)	1 (1.0)
Distribution of CIMT of study population	**Variable**	**Total Patients (n=100) (%)**
	CIMT (mm):	
	Normal (≤0.90)	69 (69.0)
	Abnormal (>0.90)	31 (31.0)
Table 4		

HTN	**n**	**CIMT (mm) Mean ± SD**		**F Value**	***P* Value**
Stage I HTN	65	0.81 ± 0.13		5.85	0.004
Stage II HTN	26	0.87 ± 0.14			
Isolated systolic HTN	9	0.94 ± 0.05			
Correlation between CIMT and HTN in Study Population (n=100)					
Comparison		**Mean Diff.**	**q Value**	***P* Value**	**95% CI of diff.**
Stage I HTN vs. Stage II HTN		-0.06	3.22	P > 0.05	0.003 to 0.141
Stage I HTN vs. Isolated Systolic HTN		-0.13	4.15	P < 0.05	0.025 to 0.247
Stage II HTN vs. Isolated Systolic HTN		-0.07	1.88	P > 0.05	0.053 to 0.188
Comparison (*P* value) of Difference in Mean CIMT Values between Three Different HTN Groups by Tukey Test					
Table 5					

The correlation between CIMT and hypertension (HTN) (stage I, stage II and isolated systolic HTN) is

summarized. The mean CIMT showed positive correlation with HTN i.e. as severity of HTN increases, mean CIMT also increases.

Comparing the mean CIMT among three HTN groups, ANOVA showed significantly ($P < 0.01$) different CIMT among the groups (F=5.85, $P = 0.004$). Further, comparing the difference in mean CIMT between the HTN groups, Tukey test showed significantly ($P < 0.05$) different and higher mean CIMT in isolated systolic HTN patients as compared to stage I HTN patients. However, it did not differ ($P > 0.05$) between stage I and stage II, and stage II and isolated systolic HTN groups i.e. found to be statistically the same.

Variable	Age	Sex	BMI	SBP	DBP	HTN	TG	TC	HDLC	LDLC	LDLC/HDLC	CIMT
Age	1.00											
Sex	0.19^{ns}	1.00										
BMI	-0.19^{ns}	-0.07^{ns}	1.00									
SBP	0.18^{ns}	-0.11^{ns}	0.04^{ns}	1.00								
DBP	0.12^{ns}	-0.19^{ns}	0.03^{ns}	0.66^{***}	1.00							
HTN	0.35^{***}	0.03^{ns}	-0.04^{ns}	0.57^{***}	0.10^{ns}	1.00						
TG	-0.21^{*}	-0.10^{ns}	0.26^{*}	-0.05^{ns}	-0.08^{ns}	-0.03^{ns}	1.00					
TC	-0.21^{*}	-0.30^{**}	0.22^{*}	0.06^{ns}	0.13^{ns}	0.05^{ns}	0.33^{**}	1.00				
HDLC	-0.32^{**}	-0.08^{ns}	-0.07^{ns}	0.06^{ns}	-0.02^{ns}	-0.05^{ns}	-0.05^{ns}	0.21^{*}	1.00			
LDLC	-0.12^{ns}	-0.22^{*}	0.29^{**}	0.15^{ns}	0.21^{*}	0.00^{ns}	0.01^{ns}	0.56^{***}	-0.04^{ns}	1.00		
LDLC/ HDLC	0.06^{ns}	-0.11^{ns}	0.30^{**}	0.04^{ns}	0.13^{ns}	-0.01^{ns}	0.10^{ns}	0.18^{ns}	-0.66^{***}	0.63^{***}	1.00	
CIMT	0.62^{***}	0.01^{ns}	0.12^{ns}	0.23^{*}	0.08^{ns}	0.33^{**}	-0.11^{ns}	0.02^{ns}	-0.34^{**}	0.15^{ns}	0.28^{**}	1.00

Table 6: Correlation (r Value) of CIMT with Demographic, Clinical and Lipid Profile in Study Population Using Pearson Correlation Analysis (n=100)

Among lipid profile, significant and positive correlation was found between TG and TC (r=0.33, $P < 0.01$), TC and HDL-C (r=0.21, $P < 0.05$), TC and LDL-C (r=0.56, $P < 0.001$) and LDL-C and LDL-C/HDL-C (r=0.63, $P < 0.001$) whereas significant and negative correlation between HDL-C and LDL-C/HDL-C (r=-0.66, $P < 0.001$). Similarly, significant and positive correlation was found between SBP and DBP (r=0.66, $P < 0.001$), and SBP and isolated systolic HTN (r=0.57, $P < 0.001$). Further, DBP also showed significant and positive correlation with LDL-C (r=0.21, $P < 0.05$).

DISCUSSION

In the present study, age of patients ranged from 30-85 years with mean (± SD) of 57.35 ± 11.68 years and median of 57 years. The highest number of patients was in the age group of ≥ 60 years. In our study, we found that the mean CIMT (± SD) of hypertensive patients (including all age groups) was 0.84 ± 0.14 mm and median was 0.84 mm. Unamba Norbert N et al. showed near similar results in which the mean CIMT among drug naive hypertensive patients was 0.83 ± 0.22mm.[15] Lakka T A et al. showed mean CIMT value as 0.77 ± 0.17 which is lower than the mean CIMT observed in the present study. This was attributed due to the atherosclerotic attenuating effect of anti-hypertensive drugs. [16]

In the present study, CIMT showed significant positive correlation with age (r=0.62, $P < 0.001$). This was concordant with the findings of Puato M et al. Gomez-Marcos M A et al. Unamba Norbert N et al. and Patel R et al.[17,18,19] Higher CIMT values are observed with age which could probably be due to the combined effect of increased blood pressure levels and aging process on the intima media.[20]

In our study, there was significant and positive (direct) correlation between age and isolated systolic hypertension (r=0.35, $P < 0.001$), and also positive but not significant correlation with SBP (r=0.18, P=0.067) and DBP (r=0.12, P=0.218).Puato M et al.[17] An increased incidence of hypertension is especially seen over the age of 50 years due to the atherosclerosis of blood vessels, contributing to hypertension.[21] Framingham heart study reported that there is an increase in hypertension in patients aged above 50 years. In the present study, there were 50 males and 50 female patients. Gender was not found to be an independent risk factor for CIMT. There was no significant correlation between CIMT and sex (r=0.01, P=0.942).[22] These are in concordance with the studies by Amar M S et al. and Yang C et al.[23,24]in with age.[25]

Similar to studies by Hussein I et al. and Ibinaiye P O et al. we found no significant difference between the right and left CIMT. [26]In the present study, the mean BMI value was 24.64 ± 2.51 and the corresponding mean CIMT value was 0.84 mm ± 0.14, a positive but non-significant correlation was observed between CIMT and BMI (r=0.12, P=0.226). These are in concordance with the studies by Amar M S et al., Yang C et al., Unamba Norbert N et al., and Khutan H et al. who reported similar positive correlation between CIMT and BMI.

Similar to a study by Baral N et al. lipid profile viz. TG (r=0.26, $P < 0.05$), TC (r=0.22, $P < 0.05$), LDL-C

(r=0.29, $P < 0.01$) and LDLC/HDLC (r=0.30, $P < 0.01$) showed significant and direct correlation with BMI indicating that increase in one may be associated with increase in other or visa-a-versa.[27]CIMT correlated positively with SBP (r=0.23, P=0.018), DBP (r=0.08, P=0.40) and isolated systolic hypertension (r=0.33, P=0.001) of all the patients, although significant correlation was seen with SBP and isolated systolic hypertension. While in our study blood pressure was measured at the time of presentation, Manios E et al. used a 24-hour SBP and DBP measurement method.[28] Ibinaiye P O et al., Naseh G et al., and Khutan H et al. showed similar results. They observed that increased CIMT had statistically significant association with duration of hypertension, high systolic BP and high diastolic BP. [29]

In hypertensive patients, high blood pressure level can cause damage to the endothelium of blood vessels with subsequent thickening of intima media complex via medial hypertrophy, a disease-related phenomenon.[30,31]This thickening of the artery wall is most likely an adaptive mechanism to compensate for the chronic increase in blood pressure levels, and it has been demonstrated in vivo and in vitro.[32] As a result, an increase in blood pressure has a significant impact on the IMT.[33]In this study, no significant correlation was observed between hypertension and lipid profile except DBP which showed significant and positive correlation with LDL-C (r=0.21, $P < 0.05$). This is in contrast to the studies done by Reddy S et al. and SarwarM S et al. which showed significant and positive correlation between hypertension and lipid profile but a negative correlation with HDL-C.[34,35] Un-fortunately there is no literature reporting the association between hypertension and lipid profile in this region of lower Assam.

CIMT showed significant and positive correlation with LDL-C/HDL-C (r=0.28, $P < 0.01$) and significant and negative correlation with HDL-C (r=-0.34, $P < 0.01$) Sengupta D et al. observed similar significant and positive correlation with LDL- C/HDL-C (r=0.664, $P < 0.001$)[36]In the Helsinki Study, LDL-C/HDL-C ratio had more prognostic value than LDL-C or HDL-C alone, a significant association between IMT and proatherogenic lipoprotein measurements was seen and the results of multiple linear regression analysis showed that the LDL-C/HDL-C ratio was the strongest predictor for IMT progression.[37]Patel R et al. showed a significant and negative correlation with HDL-C. Plenty of studiesdemonstrated HDL as the independent negative predicator of atherosclerosis as it displayed pleiotropic effects.

Recently, research was focused on HDL-based therapy in acute ischemic stroke because it may have antioxidant, anti-inflammatory, and anti-apoptotic capabilities, as well as leukocyte modulation and platelet activation properties.[38] Yang C et al. also showed the similar positive correlation with LDL-C/HDL-C (r=0.788, $P < 0.001$) and significant and negative correlation with HDL-C (r=-0.093, $P <0.001$). The ratio of LDL-C to HDL-C in the blood is an independent indicator of increased carotid IMT. Non-HDL cholesterol and lipid ratios, such as total/HDL cholesterol and LDL/HDL cholesterol ratios have been shown to have a higher predictive value for coronary atherosclerotic development or regression than traditional lipid profiles. Through oxidative stress and endothelial dysfunction, the coexistence of hypertension and dyslipidaemia has been shown to have multiplicative rather than additive effects on cardiovascular disease risk.

Dyslipidaemia and hypertension both result in arterial wall thickening, but through distinct mechanisms. Through the process of atherosclerosis, dyslipidaemia causes arterial wall thickening by oxidative modified lipoproteins being taken up by macrophages, resulting in the production of cytokines and growth factors that stimulate smooth muscle cell proliferation and secrete extracellular matrix of the characteristic fibrous tissue of the fibro fatty atheroma. Hypertension can exacerbate the atherosclerotic process by injuring the endothelium wall causing vascular cell growth. This implies that increased CIMT in hypertensive patients with dyslipidaemia is the result of a pathologic synergy.[39]

CONCLUSION

Hypertension and dyslipidaemia are an important risk factor for increasing CIMT in high risk group. Multiple regression analysis shows that among the multiple lipid profiles, the most important factor for increasing CIMT is LDL: HDL. Isolated systolic hypertension presented with significantly higher CIMT values. Therefore, patients with isolated systolic hypertension should alert physicians to perform a more thorough evaluation of the cardiovascular profile of such patients. We conclude that the results support the importance of screening hypertensive patients by carotid ultrasound Doppler to assess the ongoing atherosclerotic changes in vessel wall.

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