Original Article

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Anterior Wall Myocardial Infarction with Special Reference to Carotid Intima Media Thickness, Ankle Brachial Pressure Index, and Echocardiographic Evaluation

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Abstract

Introduction: Acute coronary syndromes, comprising unstable angina, non-ST-segment elevation myocardial infarction (MI) and ST-segment elevation MI, are the most common causes of mortality in patients with coronary artery disease (CAD), which is the leading cause of mortality and morbidity in the world.

Aims and Objectives: To study the correlation between carotid intima media thickness (CIMT), ankle brachial pressure index (ABI), echocardiography (ECHO), and anterior wall MI (AMI).

Results: CIMT was abnormal in 54.4% patients, ABI was found to be abnormal in 13.3% patients, 2D ECHO was abnormal in 91.1% patients. Hypertension, diabetes and that with family history of MI were statistically significant ($P < 0.01$), while dyslipidemia, smoking status, obesity and previous history of MI were not statistically significant ($P > 0.05$) in abnormal and normal CIMT groups. Furthermore, hypertension, smoking status, and family history of MI were statistically significant ($P < 0.05$), while diabetes, dyslipidemia, obesity, previous history of MI were not statistically significant ($P > 0.05$) in abnormal and normal ABI groups. There was statistically significant ($P < 0.05$) difference of abnormal ABI according to CIMT abnormality and also a significant difference of abnormal CIMT according to ABI abnormality. The presence of regional wall motion abnormalities and depressed left ventricular ejection fraction (LVEF) were statistically significant in abnormal and normal CIMT groups.

Conclusion: CIMT, a measure of carotid atherosclerosis, denotes generalized atherosclerosis due its correlation to MI, asymptomatic peripheral artery disease and should be used as a screening test for detecting adults at risk of CAD; while ABI does not correlate with MI and can be used in combination with CIMT for effective high-risk screening.

Key words: Ankle brachial pressure index, Carotid intima media, Myocardial infarction

INTRODUCTION

Acute coronary syndromes (ACS), comprising unstable angina (UA), non-ST-segment elevation myocardial infarction (NSTEMI), and STEMI, are the most common causes of mortality in patients with coronary artery disease (CAD), which is the leading cause of mortality and morbidity in the world.[1]

Extracranial carotid artery disease has been associated with increased prevalence of significant coronary atherosclerosis and acute coronary events.[2-5]

Carotid intima media thickness (CIMT) measurement is a surrogate marker for atherosclerosis.[6]

CIMT is closely associated with many traditional cardiovascular risk factors (such as cholesterol, diabetes, blood pressure [BP], and smoking), some new risk

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factors (such as lipoprotein, platelet aggregability, and hyperhomocysteinaemia), and target organ damages (such as left ventricular hypertrophy, microalbuminuria, and decreased ankle-brachial index [ABI]).[7]

CIMT is strongly associated with the risk of MI and stroke in asymptomatic older adults.[8]

ABI is a reliable indicator of high coronary risk[9] and is significantly related to the presence of CAD.[10]

An ABI cut-off point of 0.9 or less has been used in clinical practice and epidemiologic studies as the indicator of PAD. ABI at this level is statistically significantly associated with higher prevalence of clinical CHD, stroke, and preclinical atherosclerosis and may be indicative of generalized atherosclerosis in middle aged men.[11]

In 1993, Newman, *et al.*,[12] found that participants with an AAI <0.8 were more than twice as likely as those with an AAI of 1.0-1.5 to have history of MI, angina, congestive heart failure, stroke, or transient ischemic attack (all $P < 0.01$).[12]

Various studies have documented an inverse correlation between ABI and IMT.[11,13-16]

Echocardiography (ECHO) is commonly used to evaluate CAD.[17] It is increasingly used as a practical and reliable means of assessing both global ventricular function and regional wall motion abnormalities (RWMA) in MI.[18]

In this study, an attempt is made to study patients having anterior wall MI (AMI) and establish the correlation of CIMT, ABI, and ECHO with AMI.

Aims and Objectives

To study, the correlation between CIMT, ABI, ECHO, and AMI.

MATERIALS AND METHODS

Study Population

A total of 90 patients with AMI were recruited on admission to the intensive care unit at Jawahar Medical Foundation's Annasaheb Chudaman Patil Memorial Medical College and hospital, from October 2012 to September 2014. In this study, we included 62 males and 28 females. Patients with confirmed diagnosis of ST-elevation (71.1%) and non-ST elevation (28.9%) AMI and satisfying the inclusion and exclusion criteria were included in the study group.

Inclusion Criteria

1. Patients more than 18 years of age
2. Patients with ST-segment elevation AMI
3. Patients with non-ST-segment elevation AMI on the basis of history, examination, cardiac enzymes, electrocardiogram changes, and 2D ECHO
4. Patients willing to give informed consent.

Exclusion Criteria

1. Patients <18 years of age
2. Patients with documented prior peripheral artery disease
3. Patients not willing to give informed consent.

Clinical Data

A detailed history with clinical examination, routine blood investigations, lipid profile, and special investigation of carotid Doppler, ABI and ECHO was performed.

Carotid Doppler Examination

Color duplex ultrasound scanning was performed by a single trained sonographer using a Toshiba Nemio XG machine with 7 MHz linear-array transducer. Subjects were examined in the supine position with the subject's neck extended and the head turned 45' to the left or right.

Ultrasound scans of the right and left last distal centimeter of the common carotid artery and the bifurcation, i.e. carotid bulb, and of the first proximal centimeter of the internal carotid arteries were performed and CIMT was measured on the far (posterior) wall using computer program based calipers.

Intima media thickness is defined as the distance from the leading edge of the lumen-intima interface of the for the present study CIMT values >0.9 mm were considered abnormal.

Carotid Plaque

Any focal thickening in the carotid lumen or CIMT >1.5 mm.

Carotid Stenosis

Lumen narrowing by >50%.

ABI

BP readings were calculated in both upper limbs and lower limbs in supine position, by raising the pressure of sphygmomanometer cuff 10-20 mmHg above systolic and cuff deflated at the rate of 2 mmHg/s; pressure at which appearance of blood flow detected by 8 Mhz probe of Toshiba Nemio XG was recorded as systolic pressure. ABI was calculated as the mean of ankle systolic pressure divided by the mean of brachial systolic pressure.

For the present study CIMT values <0.9 were considered abnormal.

ECHO

2D Transthoracic ECHO was performed on Envisor-C machine using 3.5MHz probe. RWMA were identified as akinesia, hypokinesia, and dyskinesia, left ventricular ejection fraction (LVEF) was calculated by Tecoliz's method.

Statistical Analysis

Data were analyzed using SPSS 16. The categorical data were presented in frequency and percent distribution. Patients were analyzed for characteristics with reference to normal and abnormal categories of CIMT (<0.9 mm normal, >0.9 mm abnormal) and ABI (>0.9 normal, <0.9 abnormal). In between parameters, association was tested using nonparametric Pearson Chi-square test or Mann–Whitney U test. Mean values of parameters in normal and abnormal CIMT was compared using the unpaired *t*-test. The level of significance was selected at $P < 0.05$, for accepting the difference in between the parameters as significant.

RESULTS

Baseline characteristics of the study population are given in Table 1.

CIMT was abnormal in 54.4% patients with AMI, a carotid plaque was present in 32.2%, ABI was found to be abnormal in 13.3% patients, 2D ECHO was abnormal in 91.1% patients.

Hypertension, diabetes and those with family history of MI were statistically significant ($P < 0.01$) while, dyslipidemia, smoking status, obesity, and previous history of MI were not statistically significant ($P > 0.05$) in abnormal and normal CIMT groups.

Furthermore, hypertension, smoking status, and family history of MI were statistically significant ($P < 0.05$), while diabetes, dyslipidemia, obesity, previous history of MI were not statistically significant ($P > 0.05$) in abnormal and normal ABI groups.

In our study, there was statistically significant ($P < 0.05$) difference of abnormal ABI according to CIMT abnormality and also significant difference of abnormal CIMT according to ABI abnormality.

CIMT was abnormal in 100% patients and carotid plaque present in 91.7% patients with abnormal ABI. There was statistically very highly significant ($P \leq 0.001$) difference of carotid plaque presence in abnormal ABI of AMI patients.

RWMA were more in abnormal then normal CIMT of AMI patients (statistically significant, $P < 0.05$)

About 98% abnormal CIMT patients had ejection fraction within 30-44% and abnormal echo findings which were statistically significant ($P < 0.05$) difference of in normal and abnormal CIMT of AMI patients.

Regional wall motion abnormality, percentage ejection fraction and abnormal 2D ECHO findings were not statistically significant ($P > 0.05$) in abnormal and normal ABI groups (Table 2).

All 12, i.e., 100% patients with abnormal ABI and 7.7% having normal ABI had carotid stenosis of more than 50%. There was statistically very highly significant ($P < 0.001$) difference of carotid stenosis within normal and abnormal ABI in AMI patients.

Table 1: Baseline characteristics of study population

Parameter	Mean±SD (range)
Age (years)	60.9±10.2 (42-76)
BMI (kg/m^2)	28±2.53
WHR	0.86±0.09
Mean CIMT (mm)	1.06±0.37
Mean ABI	1.00±0.11
LVEF (%)	42.4±5.9
Total cholesterol (mg/dl)	218.48±26.74
LDL (mg/dl)	138.3±29.8
HDL (mg/dl)	45.26±9.54
Triglycerides (mg/dl)	182.04±79.29
Hypertension (%)	62.2
DM (%)	44.4
Dyslipidemia (%)	65.6
Smoking (%)	41.1
Obesity (%)	20.0
Family history of MI (%)	23.3

MI: Myocardial infarction, ABI: Ankle brachial pressure index, CIMT: Carotid intima media thickness, DM: Diabetes mellitus, HDL: High density lipoprotein, LDL: Low density lipoprotein, LVEF: Left ventricular ejection fraction, BMI: Body mass index, WHR: Waist to hip ratio, SD: Standard deviation

Table 2: Abnormalities of CIMT, ABI, and ECHO in study group

Parameter	Percent
CIMT	
Abnormal	54.4
Normal	45.6
Carotid plaque	
Present	32.2
Absent	67.8
ABI	
Abnormal	13.3
Normal	86.7
2D ECHO	
Abnormal	91.1
Normal	8.9

ABI: Ankle brachial pressure index, CIMT: Carotid intima media thickness, ECHO: Echocardiography

Mean CIMT showed a linear increase with increasing number of risk factors. While such a relation could not be seen with decreasing mean ABI.

There was a highly statistically significant ($P < 0.001$) difference in mean CIMT values in diabetics (0.95 ± 0.31 mm) as compared to non-diabetics (0.74 ± 0.14 mm).

All 6 patients that died were having all 3 abnormalities, i.e., CIMT, ABI and ECHO abnormal in them. While of those 12 patients having AMI with these 3 abnormalities 50% survived and 50% had died (Table 3).

DISCUSSION

As a screening test, imaging must be safe, sensitive, and affordable. Measurement of CIMT by B-mode ultrasound is non-invasive, sensitive, and reproducible technique for identifying and quantifying atherosclerotic burden and cardiovascular disease (CVD) risk.[19]

Nine published prospective studies that included at least 1000 asymptomatic participants have examined CIMT and CVD risk. Each study demonstrated that CIMT was significantly associated with risk for MI, stroke, death from coronary heart disease, or a combination of these events. In most of these studies, the ability of CIMT to predict future CVD events was independent of traditional risk factors.[19]

The ABI may help to identify asymptomatic individuals in the general population who are at increased risk of subsequent cardiovascular events. It has shown the most promise as a potential tool in clinical practice and has been most wide investigated.[20]

Su *et al.*, 2005,[21] found cardiovascular events in 50% of patients with ABI <0.9 as compared to only 7% in group with ABI ≥0.9, which was statistically very significant ($P < 0.01$).[21]

They also observed that ABI <0.9 and three-vessel CAD were significant predictors of cardiovascular events.[21]

Table 3: Number of risk factors present with their mean CIMT and ABI of patients with AMI

Number of risk factors	Frequency	Percent	Mean CIMT	Mean ABI
0	7	7.8	0.721	1.078
1	13	14.4	0.751	1.006
2	26	28.6	1.003	1.022
3	16	17.8	1.215	0.966
4	23	25.6	1.197	1.025
5	5	5.6	1.563	0.900
Total	90	100.0		

AMI: Anterior wall myocardial infarction, ABI: Ankle brachial pressure index, CIMT: Carotid intima media thickness

In our study, mean age was 60.9 ± 10.2 years (range 42-76 years). This was comparable with Kablak-Ziembicka *et al.*[22] (59.3 years), Shetty *et al.*[23] (58.72 years), Keo, *et al.*, 2011[24] (65.5 ± 9.4 years), Aljabri, *et al.*[25] (62 ± 14.3 years).

While it was not comparable with Jadhav and Kadam[26] (52.8 ± 8.7 years), and Hansa *et al.*,[5] (49.7 ± 10.5 years).

We did not find any statistically significant difference of age groups according to abnormal (>0.9 mm) and normal (≤0.9 mm) CIMT. Also, there was no significant difference of mean age in between abnormal and normal CIMT.

We did not find any statistically significant difference of age groups according to abnormal (<0.9 mm) and normal (≥0.9 mm) ABI. Furthermore, there was no significant difference of mean age in between abnormal and normal ABI.

This is not comparable to Su, *et al.*[21] and Brasileiro, *et al.*[16] who demonstrated that elderly population has a lower ABI. This is because only 34% of patients above 70 years. Were smokers in our study and as smoking is significantly related to abnormal ABI, our observations lack the association.

In our study, we did not observe any statistically significant difference of gender according to abnormal (>0.9 mm) and normal (≤0.9 mm) CIMT. Furthermore, there was no statistically significant difference in the mean values of CIMT according to gender. This is not concordant with Linhart, *et al.*,[27] who observed CIMT to be significantly increased to a greater extent in young men than young women, as their patients were <45 years males and <50 years females.

We did not observe any statistically significant difference of gender according to abnormal (<0.9 mm) and normal (≥0.9 mm) ABI. This is concordant with observations of Newman, *et al.*,[12] and Brasileiro, *et al.*, 2013.[16]

In our study, family history of CAD was present in 23.3% of patients. There was a statistically significant difference of presence of family history of CAD in patients with abnormal CIMT (81%) and abnormal ABI (28.6%).

Linhart, *et al.*, 2012,[27] found statistically significant difference of presence of history of cardiovascular events in first-degree relatives in men and women as compared with controls.

In our study, 71.1% were overweight and 20% were obese, comparable with Kablak-Ziembicka *et al.*[22] (20.3% obese), and Keo *et al.*[24] (45.3% overweight, 21.1% obese).

In our study, there was no significant difference of BMI groups according to abnormal and normal CIMT.

In our study, there was no significant difference of BMI groups according to abnormal and normal ABI, comparable to Newman *et al.*;[12] Su *et al.*;[21] Aljabri *et al.*[25] and Brasileiro *et al.*[16]

In our study, current smoking was found in 41.1% of AMI patients. It was higher than that observed by Jadhav and Kadam[26] (31.3%), Gupta Hansa, *et al.*,[5] (21%), Keo *et al.*,[24] (11.7%).

While it was lower as compared to that observed by Kablak-Ziembicka *et al.*[22] (64.4%), Shetty, *et al.*[23] (51.4%).

In our study, there was statistically significant difference of smoking in AMI patients as regards abnormal and normal ABI, comparable with Newman *et al.*;[12] Su *et al.*[21]

Papamichael *et al.*[28] found smoking ($P = 0.025$) was significantly related to ABI in the multiple regression analysis.

However, Aljabri *et al.*[25] and Brasileiro *et al.*[16] found no significant difference of ABI in between smokers and nonsmokers.

We observed 62.2% of AMI patients to be hypertensives. It was comparable with Kablak-Ziembicka AK, *et al.*[22] (62 %), Gupta Hansa, *et al.*[5] (54.5%), Shetty *et al.*[23] (52.96%) Keo, *et al.*[24] found 83.5% of CAD patients to be hypertensives.

In our study, hypertension was statistically highly significant in patients with abnormal CMIT as compared to those with normal CIMT. This is comparable to Jadhav and Kadam[26] who observed that abnormal IMT had the strongest correlation for CAD in subjects with hypertension (with an incidence of 22.2%) as against those without CAD (only 3.6%).

Linhart *et al.*[27] found hypertension 15.3% and 30% of young male and female MI survivors respectively. The lower prevalence as compared to other studies is attributed to the younger cohort in their study.

In our study, hypertension was statistically highly significant in patients with abnormal ABI as compared to those with normal ABI. However, it is not comparable with below studies.

Aljabri *et al.*[25] found hypertension in 55% of CAD patients, which was not significant as regards abnormal and normal ABI.

Brasileiro *et al.*[16] found statistically no significant difference of ABI in between patients with and without hypertension.

We found diabetes in 44.4% of AMI patients. This was comparable with Jadhav and Kadam[26] (51.5%). However, it was higher as compared to with Gupta Hansa, *et al.*, 2003[5] (31%), Keo *et al.*[24] (31.2%) and Shetty *et al.*[23] (20%).

In our study, there was a highly statistically significant difference in mean CIMT in diabetics as compared to non-diabetics. We also found statistically highly significant difference ($P < 0.001$), with higher percentage of patients (75%) having abnormal CIMT in diabetics compared to normal CIMT (25%) amongst the AMI patients.

It is comparable to the Chennai Urban Population Study[29] where mean IMT values in Diabetic subjects were significantly raised (0.95 ± 0.31 mm) compared to non-diabetic subjects (0.74 ± 0.14 mm; $P < 0.001$).

We found statistically no significant difference of diabetes in patients having abnormal ABI comparable with Aljabri *et al.*[25] and Brasileiro *et al.*[16]

While Papamichael *et al.*[28] found diabetes ($P = 0.01$) was significantly related to ABI in the multiple regression analysis.

In our study, there was no statistically significant difference of presence of dyslipidemia or individual lipid abnormalities in between abnormal and normal CIMT.

In our study, there was no statistically significant difference of presence of dyslipidemia or individual lipid abnormalities in between abnormal and normal ABI. These observations are comparable with Su *et al.*;[21] Aljabri *et al.*;[25] and Brasileiro *et al.*[16]

In our study, mean CIMT increased with the increasing number of risk factors with mean IMT being highest in patients with 5 risk factors. This finding is concordant with Atherosclerosis Risk in Communities study,[30] and Gupta Hansa, *et al.*[5]

CIMT: In our study, CIMT was abnormal in 54.4% of AMI patients. This was comparable to Jadhav and Kadam[26] (59.2%) and not comparable to Brasileiro, *et al.*[16] (69.5%) and Liu *et al.*[7] (77.78%).

In our study, mean CIMT was 1.06 ± 0.37 mm which was comparable with Simons *et al.*[31] (0.94 ± 0.33 mm), Shetty, *et al.*[23] (0.923 ± 0.123 mm) and not comparable to Gupta Hansa, *et al.*[5] (0.82 mm) Visonà *et al.*[32] (1.45 ± 0.95 mm).

We found, CIMT was increased in both ST elevation and non ST elevation MI, but the difference was not statistically significant.

We found carotid plaque in 32.2 %, which is comparable to study by Salonen and Salonen,[6] who found small plaques in 30% subjects. It is not comparable to Sirimarco *et al.*[33] (44%).

We observed very highly statistically significant relation of patients with carotid plaque with abnormal ABI. Similarly, presence of carotid stenosis was very highly statistically significant in patients with abnormal ABI. This is comparable to Newman *et al.*[12] Ogren *et al.*[34]

ABI

In our study, ABI was abnormal in 13.3% of patients, which is comparable with Newman *et al.*[12] who found abnormal ABI in 13.8% males and 11.4% females.

In our study, mean ABI was 1.00 ± 0.11 in patients with anterior wall myocardial infarction. Mean ABI of those with ABI <0.9 was 0.81 ± 0.03. This was comparable to Su *et al.*[21]

ABI and CIMT

In our study, there was statistically significant difference of abnormal ABI according to CIMT abnormality and also significant difference of abnormal CIMT according to ABI abnormality. This is concordant with Zheng *et al.*;[11] Allan *et al.*;[13] Simons *et al.*;[14] Sodhi *et al.*;[15] Papamichael *et al.*;[28] and Brasileiro *et al.*[16]

In our study, 2D ECHO was abnormal in 91.1% of patients. There was statistically significant difference of ECHO abnormalities of RWMA, and LVEF, being more in abnormal CIMT group. As ECHO is a proven definitive test for detecting MI, our above finding suggests CIMT is correlated to AMI patients.

Horowitz *et al.*[18] observed 94% patients with clinical AMI had RWMA on the initial 2D ECHO.

In our study, RWMA were seen in patients as akinesia (31.1%), dyskinesia (31.1%), and hypokinesia (28.9%).

Chizynski *et al.*[35] observed diffuse hypokinesis in 38%, regional akinesis in 29%, and regional dyskinesis in 33% with impaired dilated left ventricular systolic and diastolic function in patients with ST elevation MI. While in Non ST elevation MI, diffuse hypokinesis in 42%, regional anterior wall hypokinesis with the normal function of other walls in 10%, and regional anterior wall akinesis with the diffuse hypokinesis of other walls in 48% was observed.

In 2004, Kablak-Ziembicka *et al.*[22] found highly statistically significant ($p<0.001$) 70.4% of CAD patients with RWMA, as compared to non-CAD.

In our study, LVEF was <45% in 91.1%, and mean LVEF was 42.4 ± 5.9%. This is comparable to McClements *et al.*[36] who in their study observed the mean LVEF significantly lower in anterior than in inferior MI (44.8% ± 11.5% vs. 53% ± 8.6%; $P = 0.001$).[36]

While, it was not comparable to, Chizynski *et al.*[35] observed LVEF range between 10 and 36% (mean: 24%) in patients with ST elevation MI, and 19-47% (mean 32%), in patients with non ST elevation MI.

In our study, we observed that 6.7% patients with AMI met with fatal outcome within the hospital stay. Of those who died, all 6 (6.7%) patients had abnormal CIMT, ABI, and ECHO findings. This is comparable to Sirimarco *et al.*[33] who observed 8.2% mortality in persons with carotid atherosclerosis, which was highly statistically significant ($P < 0.0001$) as compared to that without.[33]

Heald *et al.*[20] observed a low ABI (<0.9) was associated with an increased risk of subsequent all-cause mortality and cardiovascular mortality after adjustment for age, sex, conventional cardiovascular risk factors and prevalent CVD.[20]

CONCLUSION

In our study, mean CIMT correlates with number of risk factors. While, mean ABI does not correlate with number of risk factors. CIMT correlates with abnormalities seen on ECHO. As ECHO is proven diagnostic test of MI, we conclude that CIMT correlates with AMI. CIMT and ABI correlate with the in-hospital mortality of MI. CIMT correlates with ABI in AMI patients. ABI does not correlate with ECHO abnormalities. As ECHO is proven diagnostic test of MI, we conclude that ABI does not correlate with AMI. CIMT, a measure of carotid atherosclerosis, denotes generalized atherosclerosis due its correlation to MI, asymptomatic peripheral artery disease and should be used as a screening test for detecting adults at risk of CAD; while ABI does not correlate with MI and can be used in combination with CIMT for effective high-risk screening.

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