

Carotid Artery Doppler Study in Patients of Myocardial Infarction and Its Correlation with Other Atherosclerotic Risk Factors

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Abstract: Atherosclerotic cardiovascular disease (CVD) is a diffuse disease involving mainly the arterial circulations of the heart, brain, kidneys and limbs. This process begins in childhood and progresses over decades. In the Framingham study, the lifetime risk for coronary artery disease in adults at age 40 was 49% for men and 32% for women.¹ The world Health organization has estimated that by 2020, the global number of deaths from CAD will be increased from 7.2 million in 2002 to 11.1 million in 2020.² Therefore the present study was undertaken to know the relationship of carotid Doppler study findings (carotid intima-medial thickness, plaques, degree of stenosis) with various risk factors for atherosclerosis in myocardial infarction patient. Also, the carotid Doppler findings were compared with severity of coronary artery disease. To evaluate the association of atherosclerotic risk factors like diabetes mellitus, Hypertension, dyslipidemia, Obesity, smoking with Carotid intima-medial thickness, plaques, degree of stenosis in carotid arteries in patients of myocardial infarction. To compare carotid Doppler findings with coronary angiographic findings in patients of myocardial infarction. The present study entitled "carotid artery Doppler studies in patients of myocardial infarction and its correlation with other atherosclerotic risk factors" was carried out at tertiary care hospital during period of January 2015 to September 2017. The present study conducted at a tertiary health care on 50 cases admitted with acute myocardial infarction. Out of 50 study subjects 30 had STEMI and 20 had NSTEMI. In Portuguese study¹¹ STEMI was present in 69% and NSTEMI in 31% myocardial infarction. His study group had 68% of STEMI and 32% of NSTEMI patients. Acute MI was seen more frequently in males as compared to females. The affected individuals were mostly in the 50-60 years age group. STEMI was more common than NSTEMI.

Keywords: Biochemical- Troponin T (qualitative assay) at the initial diagnosis of AMI and later repeated after 12 hrs. If initially is negative. It was done using troponin T (NAC) act kit using immunoinhibition/ modified IFCC method using Micro lab 200 Merck analyzer. Electrocardiogram-A standard 12 lead ECG was obtained on admission and whenever post infarction angina occurred. ECG was done using BPL single channel ECG machine.

INTRODUCTION

Atherosclerotic cardiovascular disease (CVD) is a diffuse disease involving mainly the arterial circulations of the heart, brain, kidneys and limbs. This process begins in childhood and progresses over decades.

Although many generalized or systemic risk factors predispose to its development, atherosclerosis affects various regions of the circulation preferentially and yields distinct clinical manifestations depending on the particular circulatory bed affected.

Atherosclerosis of the coronary arteries commonly causes ischemic heart diseases; atherosclerosis of the arteries supplying the central nervous system frequently causes cerebro-vascular accidents. In the peripheral circulation, atherosclerosis causes intermittent claudication and gangrene and can jeopardize limb viability. Involvement of the splanchnic circulation can cause mesenteric ischemia. In the Framingham study, the lifetime risk for coronary artery disease in adults at age 40 was 49% for men and 32% for women [1].

The world Health organization has estimated that by 2020, the global number of deaths from CAD will be increased from 7.2 million in 2002 to 11.1 million in 2020[2] various methods have been used since time to measure the degree of atherosclerosis, one of them being the carotid Doppler studies. The intima media thickness of the carotid artery (CIMT), presence of plaques and degree of stenosis can be measured with a high degree of accuracy and reproducibility by carotid Doppler sonography which provides a reliable and valid estimate of the degree of atherosclerosis in carotid arteries.

Therefore the present study was undertaken to know the relationship of carotid Doppler study findings (carotid intimo-medial thickness, plaques, degree of stenosis) with various risk factors for atherosclerosis in myocardial infarction patient. Also, the carotid Doppler findings were compared with severity of coronary artery disease.

Once the strongest correlates and associates of changes in carotid artery are uniformly unraveled in patients of myocardial infarction, we would have immense modifying power in our hands to prevent atherosclerotic vascular events.

AIMS AND OBJECTIVES

- To evaluate the association of atherosclerotic risk factors like diabetes mellitus, Hypertension, dyslipidemia, Obesity, smoking with Carotid intimomedial thickness, plaques, degree of stenosis in carotid arteries in patients of myocardial infarction.
- To compare carotid Doppler findings with coronary angiographic findings in patients of myocardial infarction.

REVIEW OF LITERATURE

Atherosclerosis is a disease of arteries and arterioles characterized by endothelial dysfunction, smooth muscle proliferation and migration, vascular inflammation, and the buildup of lipids, cholesterol, calcium and cellular debris within the intima of the vessel wall, This build up results in plaque formation, vascular remodeling, acute and chronic luminal obstruction, abnormalities of blood flow and diminished oxygen supply to target organs [3]. Atherosclerosis-Atherosclerosis (ath" er-o-skleh-ro'sis) comes from the greek word athero (meaning-gruel or paste) and sclerosis (meaning-hardness)[12].

Pathogenesis-The sequential components of the "response to injury" theory are as follows[3]- Endothelial injury-Which causes increased vascular permeability, leukocyte adhesion and thrombosis-Accumulation of lipoproteins mainly LDL and its oxidized form in vessel wall.

Dyslipidemia-It is well- established that the risk of cardiovascular disease and mortality from coronary artery disease is directly correlated with the concentration of cholesterol in blood. More specifically the level of serum LDS (low density lipoproteins) has a major role as it is transported to peripheral tissues. Serum HDL (high density lipoproteins) on the other hand mobilizes cholesterol from the tissues to liver for excretion in bile thus it correlates with reduced risk [4].

Hypertension-A major risk factor atherosclerosis. Angiotensin- II, a peptide involved in development of hypertension besides having vasoconstrictor properties can instigate intimal inflammation. For example, angiotensin-II elicits the production of superoxide anion, a reactive oxygen species, from arterial endothelial cells and smooth muscle cells (SMCs). Angiotensin II can also increase the expression of proinflammatory cytokines such as interleukin (IL)-6 and MCP-1 (Monocyte chemoattractant protein-1) by arterial SMCs and the leukocyte adhesion molecule VCAM-1 by endothelial cells. All these factors accelerate the progression of development of atherosclerosis.

Diabetes Mellitus-Diabetes is yet another risk factor for atherosclerosis of high importance. Many theories have been put but 3 distinct metabolic pathways have been implicated in the deleterious effect of persistent hyperglycemia on peripheral tissues and hence accelerating atherosclerosis. Formation of advances glycation end products. Activating of protein kinase C. Increased production of polyols. These all mechanism accelerates the rate of atherosclerosis [5].

Smoking-Smoking also accelerates the process of atherosclerosis. In smokers there is increase free radical formation which causes endothelia injury. Obesity-Obesity not only predisposes to insulin resistance and diabetes, but also leads to atherogenic dyslipidemia. The resulting elevation in VLDL can lower HDL cholesterol by augmenting exchange from HDL to VLDL by cholesteryl ester transfer protein, thus accelerating atherosclerosis [6].

Diet-Diet rich in Saturated fats like eggs, meat, milk etc, tend to raise plasma cholesterol level and predispose to atherosclerosis. On the contrary, a diet rich in poly-unsaturated fats. Age-Age has a dominant influence on progression of atherosclerosis. As the age advances there is progressive increase in thickness and stiffness of arterial wall.

Sex-Incidence and severity of atherosclerosis is more in men than in women. Prevalence of atherosclerotic IHD is about three times higher in men in fourth decade than females. This may be due to protective effect of estrogens [7]. Novel risk factors-Recent studies have shown that a number of factors besides traditional risk factors are implicated in

development and progress of atherosclerosis. These are C- reactive protein, elevated level of homocystine, lipoprotein (a) and factors affecting hemostasis.

Compared with CIMT measurement, the major advantage of CMR is the ability to directly image the coronary arteries and perhaps characterize unstable plaques [8]; the major disadvantage is the high cost of the study compared with ultrasonography.

Common carotid artery-The right and left carotid arteries differ in length and often, the right carotid, originates from the brachiocephalic trunk behind the right sternoclavicular joint. The left carotid originates directly from the aortic arch immediately posterolateral to the brachiocephalic trunk.

External carotid Artery- The external carotid artery begins lateral to the upper border of thyroid cartilage, at the level of the disc between the third and fourth cervical vertebrae. Internal carotid artery –The internal carotid artery supplies most of the ipsilateral cerebral hemisphere, eye, forehead and the nose. The internal carotid artery can be divided into cervical, petrous, cavernous and cerebral parts.

MATERIALS AND METHODS

The present study entitled carotid artery Doppler studies in patients of myocardial infarction and its correlation with other atherosclerotic risk factors” was carried out at tertiary care hospital during period of January 2015 to September 2017.

Study design: Descriptive study

Study Setup

Intensive care unit and Radio diagnosis, Cardiology Department of tertiary care Hospital.

Sample size: 50 cases of acute myocardial infarction were included in the present study.

Inclusion criteria-Patients suffering from acute myocardial infarction and those giving a written valid consent to participate in study

Exclusion Criteria-Previous H/O of acute myocardial infarction, Previous H/O CVA, Rheumatic heart diseases, Connective tissue diseases, Acute or chronic kidney disease.

Investigations

Biochemical- Troponin T (qualitative assay) at the initial diagnosis of AMI and later repeated after 12 hrs. If initially was negative. It was done using trop T (NAC) act kit using immunoinhibition/ modified IFCC method using Micro lab 200 Merck analyzer. Electrocardiogram-A standard 12 lead ECG was obtained on admission and whenever post infarction angina occurred. ECG was done using BPL single channel ECG machine.

Coronary angiography-All patients were referred to Cardiology Department for coronary angiography (CAG). It was carried out in most patients of myocardial infarction if they could afford it. Stenosis of 70% or greater of the arterial intraluminal diameter of the right coronary artery and left anterior descending or circumflex branches of the left coronary artery were considered significant. Single vessel disease (SVD), double vessel disease (DVD) and triple vessel disease (TVD) were defined as significant stenosis of one, two and three coronary arteries respectively. The remaining which did not have 70% or greater stenosis in any of the arteries was labeled as non significant CAD.

RESULTS

Table-1: showing age distribution

Total No	MIN. age in yrs.	Max. age in yrs.	Mean	S.D
50	42 yrs.	65yrs	55.3 yrs.	6.2

Above table shows the age distribution of patients. Mean age was 55.3 yrs

Table-2: Showing gender distribution

Gender	Number of patients	Percentage
Male	41	82%
Female	9	18%
Total	50	100%

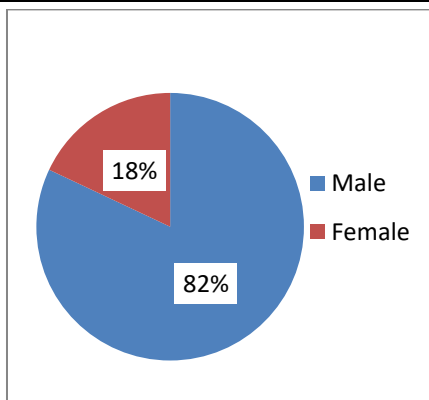


Fig-1: Chart showing gender distribution

Above chart shows that out of total 50 patients 41 were males and 9 were females (Fig-1). Above table shows that most common symptom in MI patient was

chest pain (98%) followed by seating (20%). None of the patient had signs of heart failure like raised JVP or basal crepitations (Table-3).

Table-3: Symptoms and Signs

Symptom/sign	Present in number of patients (n=50)	Percentage
Chest pain	49	98%
Breathlessness	4	8%
Palpitation	8	16%
Seating	10	20%
Nausea	4	8%
Vomiting	4	8%
Tachycardia	31	62%
Raised JVP	0	0%
Basal crepitations	0	0%

Table-4: Showing various risk factors among MI patients

Risk factors	Number of patients	Percentage
Smoking	16	32%
Obesity	26	52%
Diabetes Mellitus	37	72%
Hypertension	34	68%
Dyslipidemia	27	54%

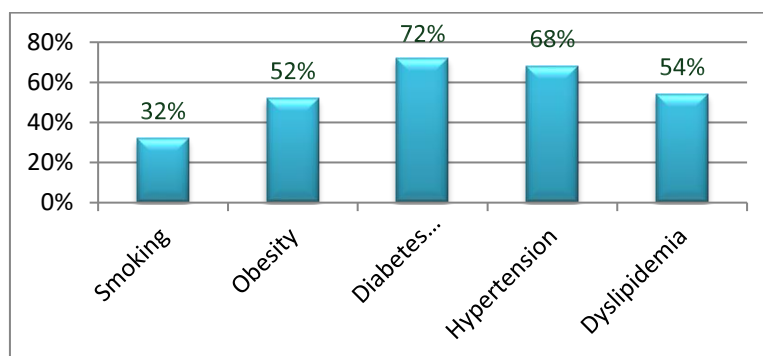


Fig-2: Chart showing various risk factors among MI patients

Table-5: Showing the diagnosis of the patients

Diagnosis	Frequency	Percentage
STEMI	30	60%
NSTEMI	20	40%
TOTAL	50	100%

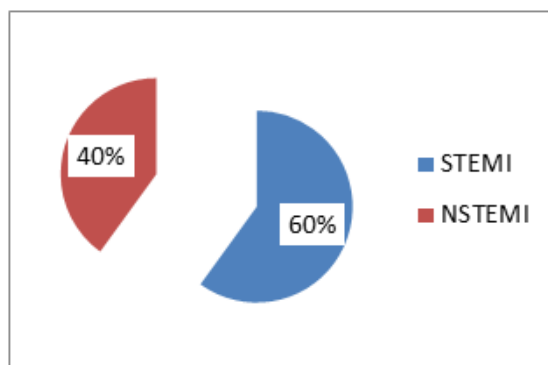


Fig-3: Chart showing the diagnosis of the patients

Above table shows that out of 50 patients 30 had STEMI and 20 had NSTEMI.

Carotid doppler findings

Table-6: showing distribution of CIMT in patients

Number of patients	Minimum CIMT in mm	Maximum CIMT in mm	Mean	S.D.
50	0.7	1.4mm	1.023	+/-0.675

Table-7: Showing no. of patients with normal and raised CIMT

Total Number of patients	Patients with CIMT upto 0.8mm	Patients with CIMT more than 0.8 mm
50	9	41

Table-8: Showing no. of patients with No, 1,2,3,4 or more plaques

Number of plaque	No plaque	Single	Two	Three	Four or more
Number of patients	15	14	7	3	1

Correlation of carotid Doppler findings with atherosclerotic risk factors

Out of all the atherosclerotic risk factors age, waist circumference, SBP, DBP, HbA1c, serum triglycerides, LDL HDL, LDL/HDL ratio were continuous variables. Linear regression analysis was applied between mean CIMT and these risk factors. Mean CIMT was taken on Y axis and the various risk factors were taken on X axis Pearson correlation coefficient (r), and the p value was calculated. P value less than 0.05 was statically significant and less than 0.01 was highly significant. The correlation coefficient

ranges from -1 to +1. A correlation of 0 to 0.25 indicates no linear association.

The sign of the Pearson correlation coefficient (r) provides important information. If the correlation coefficient is positive, high values of one variable are associated with high values of the other variable. If the correlation coefficient is negative, low values of one variable are associated with high values of the other variable. Smoking and gender being a binomial data ie either (yes/no) or (male/female) respectively were analyzed separately for correlation with mean CIMT by applying independent sample t test.

Table-9: Showing no of plaques vs coronary angiographic findings

Angiography	Number of Plaques in carotid					Total
	No Plaque	Single	Two	Three	Four or more	
Non-Significant	9	0	0	0	0	9
SVD	6	13	4	2	0	25
DVD	0	1	7	3	2	13
TVD	0	0	0	2	1	3
Total	15	14	11	7	3	50

Above table shows the number of plaques in non-significant, SVD, DVD, TVD, 10 patients had a non-significant CAD on coronary angiography all of these had no stenosis in carotids. Total number of patients having no stenosis in carotids was 30. So, no stenosis in carotid had a positive predictive value of

33.3% for having a non-significant disease on coronary angiography.

DISCUSSION

The present study conducted at a tertiary health care on 50 cases admitted with acute myocardial

infarction. Age distribution-Mean age of patients was 55.3 years, it is comparable to study done by Kablak-Ziembicka A *et al.* [9] in their study mean age group was 58.8 years Agrawal *et al.*¹⁰ conducted a similar study in which the mean age of study population was 59.7 years. Gender Distribution- out of 50 cases 41 (82%) were male and 8 (18%) were female. Study done by Agrawal *et al.* [10] there were 71% and 29% fe,a;es/

Symptoms and signs- Chest pain (98%) was most common symptom in patients with MI followed by sweating (20%) Palpitation (16%), Dyspnea (8%), Nausea and vomiting (8%). In the study done by Henriksson *et al.* [13] most common symptoms were chest pain 68% followed by shortness of breath (48%). Chest pain is the most common and a classical symptom of myocardial infarction it was absent in only one of the study subject, who was severely uncontrolled diabetic with HbA1c None of the patient had signs of heart failure like raised JVP or basal crepitation's.

Clinical Diagnosis- out of 50 study subjects 30 had STEMI and 20 had NSTEMI. In Portuguese study [11] STEMI was present in 69% and NSTEMI in 31% myocardial infarction. His study group had 68% of STEMI and 32% of NSTEMI patients.

SUMMARY

- 50-60 years was the most common age group affected with Acute MI.
- 82% patients were males and 18% were females.
- Chest pain was present in 98% of patients.
- STEMI (66%) was more common type of acute MI than NSTEMI (33%).
- Out of 50 patients 32% were smokers, 52% were obese, and 68% were Hypertensive, 72% were diabetic, 54% had dyslipidemia.
- Diabetes mellitus was most common risk factor present in study.
- Smoking (p value 0.049) was minimally correlating with increased CIMT.

CONCLUSION

- Acute MI was seen more frequently in males as compared to females.
- The affected individuals were mostly in the 50-60 years age group.
- STEMI was more common than NSTEMI.
- Uncontrolled diabetes mellitus is most important risk factor for degree of carotid atherosclerosis in MI patients.
- Amongst the serum Triglycerides, serum LDL, Serum HDL and LDL/HDL ratio raised levels of serum HDL was most significant negative factor for carotid atherosclerosis and raised levels of serum triglyceride was most significant positive factor for carotid atherosclerosis.
- CIMT correlated well with severity of coronary artery disease.

- Number of plaques also correlated with coronary artery disease severity.

Limitations of study-Our study sample was small and confined to one tertiary car hospital so large scale studies are required for its definite role in clinical practice. CIMT being a reflector of cumulative atherosclerosis burden, it does not help to find out the individual contribution of multiple etiological factors for atherosclerosis.

Applications of study-In patients of MI with multiple risk factors, carotid sonography can be useful measure for risk stratification. It can be a very useful tool as it is noninvasive, reproducible, ease of application, low cost and has a high correlation with atherosclerotic risk factors.

REFERENCES

1. Lloyd-Jones DM, Larson MG, Beiser A, Levy D. Lifetime risk of developing coronary heart disease. *The Lancet.* 1999 Jan 9;353(9147):89-92.
2. Bonow RO, Smaha LA, Smith SC, Mensah GA, Lenfant C. World Heart Day 2002: the international burden of cardiovascular disease: responding to the emerging global epidemic. *Circulation.* 2002 Sep 24;106(13):1602-5.
3. Kumar V, Abbas AK, Aster JC. Robbins Basic Pathology E-Book. Elsevier Health Sciences; 2017 Mar 8.
4. Gau GT, Wright RS. Pathophysiology, diagnosis, and management of dyslipidemia. *Current problems in cardiology.* 2006 Jul 1;31(7):445-86.
5. Anirban maitra. Pathological basis of disease by robbins and cotran. 8th edition pg no 1138-1140.
6. Mohan H. The Blood Vessels and Lymphatics. Harsh Mohan. Text book of Pathology. New Delhi: Jaypee Brothers Medical publishers. 1995:239-50.
7. Sweitzre N Douglas P. Cardiovascular disease in women, Braunwald heart disease, 2005 7th edition:1951.
8. Cohn JN, Quyyumi AA, Hollenberg NK, Jamerson KA. Surrogate markers for cardiovascular disease. *Circulation.* 2004 Jun 29; 109(25 suppl 1):IV-31.
9. Kablak-Ziembicka A, Tracz W, Przewlocki T, Pieniazek P, Sokolowski A, Konieczynska M. Association of increased carotid intima-media thickness with the extent of coronary artery disease. *Heart.* 2004 Nov 1;90(11):1286-90.
10. Agarwal AK, Gupta PK, Singla S, Garg U, Prasad A, Yadav R. Carotid intimomedial thickness in type 2 diabetic patients and its correlation with coronary risk factors. *JAPI.* 2008 Aug 5;56:581-6.
11. Portuguese journal of cardiology. 2010 29 (6) 947-955.
12. Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo. Harrison's principles of internal medicine. New York: Mc Graw-Hill Medical Publishing Division. 2008; 18th edition: 1987.

13. Henriksson C. Coronary heart disease and early decision making, from symptoms to seeking treatment. Sweden: Uppsala University; 2011. pp: 11-41.