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A Study of Clinical Profile, Prevalence of Risk Factors and Assessment of Microvascular Dysfunction by Coronary Angiography in Patients of Ischemic Heart Disease Admitted in the Department of Cardiology of a Tertiary Care Hospital in Kolkata

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ABSTRACT

BACKGROUND Substantial research and clinical evaluation has been directed towards the understanding, diagnosis and management of coronary artery disease. It is also a well-known fact that myocardial ischaemia may occur in the absence of overt atherosclerotic diseases in the epicardial coronary vessels as demonstrable by coronary angiography. Contrary to the epicardial coronary vasculature, the coronary microcirculation has remained elusive to conventional imaging techniques.

AIMS AND OBJECTIVES:

1. To assess the severity and pattern of microvascular dysfunction in patients with ischemic heart disease by corrected TIMI Frame Count (CTFC) and Myocardial Blush Grading (MBG) in vessels without any flow limiting disease.
2. To study the correlation between microvascular dysfunction and various risk factors for CAD

Methods – We measured CTFC in 400 patients. Myocardial blush in non-infarct territory was also studied. Correlation between various atherosclerotic risk factors and CTFC was studied. We also compared the CTFC between patients with different types of presentations

Results- We found that coronary flow was significantly slower in non infarct related arteries in the setting of STEMI and NSTEMI despite no obstructive epicardial coronary lesion as compared to coronary flow in the absence of acute MI ($p < 0.001$). CTFC in males was more than in females but was not statistically significant ($p > 0.10$). Also, significant slow flow was present in Non infarct related arteries in STEMI as compared to NSTEMI. Similarly significant slow flow was present in the patients with unstable angina when compared with patients presenting with stable angina. Among patients with STEMI and NSTEMI, significant slow flow was present in the infarct related artery in STEMI patients. The amount of microvascular dysfunction was directly related to number of atherosclerotic risk factors which again suggests that microvascular dysfunction might precede epicardial thrombosis. Diabetes and dyslipidemia predicted higher CTFC.

Keywords: microvascular dysfunction, corrected TIMI Frame Count (CTFC), Myocardial Blush Grading (MBG), coronary artery disease

INTRODUCTION

The clinical significance of coronary microvascular dysfunction (CMVD) has not been given as much attention as epicardial CAD. Microvascular dysfunction has also been documented in patients with obesity, diabetes, hypercholesterolaemia, hypertension, and in smokers in the absence of epicardial CAD.

Coronary microvascular function may also be impaired in patients with epicardial CAD in the non-stenotic vascular bed. Several diagnostic techniques have been employed to evaluate tissue-level microvascular perfusion. Studies using myocardial contrast echocardiography, magnetic resonance, Doppler flow wire, nuclear imaging, myocardial blush, corrected TIMI frame count, and TIMI myocardial perfusion grade, have provided investigators with information on the incidence of microvascular dysfunction and its clinical consequences in patients with abnormal myocardial perfusion at the tissue level.[1,2]

The objective of this study is to represents the clinical profile, prevalence of risk factors and assessment of microvascular dysfunction by coronary angiography in patients of ischemic heart disease admitted in the department of cardiology of a tertiary care hospital.

AIMS AND OBJECTIVES:

1. To assess the severity and pattern of microvascular dysfunction in patients with ischemic heart disease by corrected TIMI Frame Count (CTFC) and Myocardial Blush Grading (MBG) in vessels without any flow limiting disease.
2. To study the correlation between microvascular dysfunction and various risk factors for CAD

MATERIALS & METHODS

1. SAMPLE SIZE-

Estimated sample size: sample consists of approximately 400 cases who are to undergo coronary angiography in the department of cardiology.

2. SAMPLE DESIGN-

Consecutive patients fulfilling inclusion criteria will be included. We will perform, corrected TIMI frame count and myocardial blush grading among ischemic heart disease patients undergoing coronary angiography in our catheterization laboratory. Before catheterization, a protocol-based clinical examination will be used to determine demographic profile, cardiac history, atherogenic risk factors, features of extra coronary vascular disease and co-morbidities

Inclusion criteria

Patients who need to undergo coronary angiography for evaluation and management of ischemic heart disease. Written informed consent will be taken from all patients.

Exclusion Criteria:

Known or suspected acute or chronic renal failure
History of contrast nephropathy
Electrolyte imbalance
Patients with severe anaemia
Hemodynamically unstable
Patients with bypass graft lesions, with heavily calcified lesions, and ostial lesions

3. STUDY DESIGN--

Hospital based Cross-sectional, observational, study

PARAMETERS TO BE STUDIED AND STUDY TOOLS

Age and Sex distribution of study population.

Major Risk Factors of atherosclerosis namely, smoking, hypertension, diabetes mellitus, dyslipidemia, family history of premature coronary artery disease.

Clinical features suggestive of atherosclerotic coronary artery disease.

Routine blood examination like hemoglobin, ESR, total WBC count, differential WBC count, platelet count.

Blood biochemistry like fasting and postprandial blood sugar, urea, creatinine, sodium, potassium, lipid profile.
Electrocardiography
Echocardiography
Tread mill test
Coronary angiography – corrected TIMI frame count and MBG

STUDY TECHNIQUES & PLAN FOR ANALYSIS OF DATA- Between the above-mentioned study period, we will screen all IHD patients undergoing coronary angiography at our hospital and evaluate them for study inclusion according to predetermined inclusion and exclusion criteria. Before catheterization, a protocol-based clinical examination will be performed to record information including demographic profile, cardiac history, indications for coronary angiography and atherogenic risk factors. CTFC and MBG will be assessed in non infarct related vessels with no flow limiting disease.

The information obtained from this study will be tabulated in a master chart and then analyzed to obtain measurable data that may be statistically analyzed, using standard methods like mean, median, standard deviation, frequency, coefficient of correlation and dispersion. These data will also be expressed through graphs and figures to convey appropriate statistical information.

PARAMETERS STUDIED AND STUDY TOOLS:

Age and Sex distribution
History and clinical examination
Assessment of major atherosclerotic risk factors including-

1) History of past or present smoking
2) Hypertension – History or on drug treatment or resting B.P. ≥ 140/90 mm Hg on at least two occasions in absence of pain, dyspnea or any other discomfort
3) Diabetes mellitus - on drug treatment or FBG ≥ 126 mg/dl, RBS ≥ 200mg/dl, PPBS ≥ 200 mg/dl or HbA1c ≥6.5
4) Dyslipidemia - history or on drug treatment or impaired lipid profile which includes either of total cholesterol≥200 mg/dl, LDL cholesterol≥130 mg/dl, HDL cholesterol ≤ 40 mg/dl, Triglycerides ≥150 mg/dl
5) Family history of premature coronary artery disease. (h/o CAD in first degree relatives before age of 55 years in males and before age of 65 years in females.)
6) Blood biochemical parameters including

1) Hemogram (haemoglobin, toatal and differential leukocyte count, platelet count, ESR)
2) Serum creatinine
3) Fasting blood sugar, Random blood sugar, post prandial blood sugar, HbA1c
4) Lipid profile
5) Serum electrolytes (Na^+, K^+)
6) Cardiac Biomarkers (CKMB, cardiac troponin)

Electrocardiography
Echocardiography
Coronary angiography

OBSERVATIONS AND RESULTS

I) TYPE OF PRESENTATION

Total number of patients studied- 400

TABLE 1- Shows the type of presentation of IHD patients

TYPE OF PRESENTATION	NUMBER OF PATIENTS
CSA	99
STEMI	212
NSTEMI	53
UA	36

II) GENDER DISTRIBUTION

TABLE 2 – GENDER DISTRIBUTION

GENDER	NUMBER OF PATIENTS
MALE	294
FEMALE	106

TABLE 3-GENDER DISTRIBUTION AMONG DIFFERENT PRESENTATION

	GENDER DISTRIBUTION			
	CSA	**UA**	**STEMI**	**NSTEMI**
MALE	66	42	164	22
FEMALE	33	11	48	14

COMPARISON OF CTFC BETWEEN MALES AND FEMALE'S PATIENTS

TABLE 4 - COMPARISON OF CTFC BETWEEN MALES AND FEMALE'S PATIENTS

GENDER	MEAN CTFC
MALE	25.96
FEMALE	25.30

TABLE 5- COMPARISON OF CTFC IN DIFFERENT PRESENTATIONS AMONG MALE AND FEMALE PATIENTS

GENDER	CTFC			
	CSA	**UA**	**STEMI NIRA**	**NSTEMI NIRA**
MALE	22.48	24.30	28.70	25.59
FEMALE	22.67	24.01	28.03	24.97

The table above shows comparison of mean CTFC between males and females. The difference was not statistically significant, $p>0.05$

III) DISTRIBUTION OF RISK FACTORS

TABLE 6- DISTRIBUTION OF RISK FACTORS IN PATIENT POPULATION

RISK FACTOR	No. OF PATIENTS WITH RISK FACTOR
DIABETES	112
HYPERTENSION	189
SMOKING	207
DYSLIPIDEMIA	251
FAMILY HISTORY OF PREMATURE CAD	43

Above TABLE shows distribution of risk factors in patients. Dyslipidemia was the most common risk factor followed by smoking, hypertension, diabetes and positive family history in that order.

NUMBER OF RISK FACTORS IN PATIENT POPULATION

TABLE 7 – DISTRIBUTION OF PATIENTS ACCORDING TO NUMBER OF RISK FACTORS

NUMBER OF RISK FACTORS	NUMBER OF PATIENTS
0	26
1	121
2	145
3	64
4	31
5	13

TABLE 7 – Shows the number of risk factors in the patients. 30% of patients had single risk factor, 36% had 2 risk factors, 16 % had 3 risk factors, 8 % had 4 risk factors and 3 % had all the risk factors.

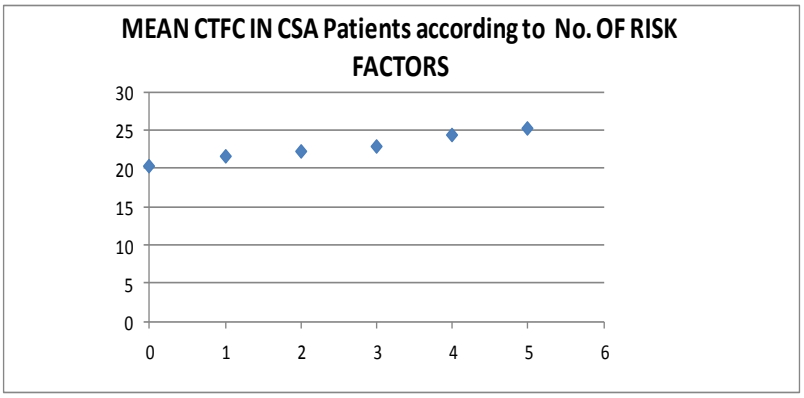
CHART 5 – DISTRIBUTION OF PATIENTS ACCORDING TO NUMBER OF RISK FACTORS

CORRELATION BETWEEN NUMBER OF RISK FACTORS AND MEAN CTFC IN CSA PATIENTS

TABLE 8 - CORRELATION BETWEEN NUMBER OF RISK FACTORS AND MEAN CTFC IN CSA PATIENTS

NUMBER OF RISK FACTORS	MEAN CTFC IN CSA PATIENTS
0	20.765
1	21.55
2	22.27
3	22.79
4	24.33
5	25.12

CHART 1 – Shows correlation between number of risk factors and mean CTFC in CSA Patients. There was a strong correlation between no. of risk factors and mean CTFC with correlation coefficient of 0.95 and p<0.001



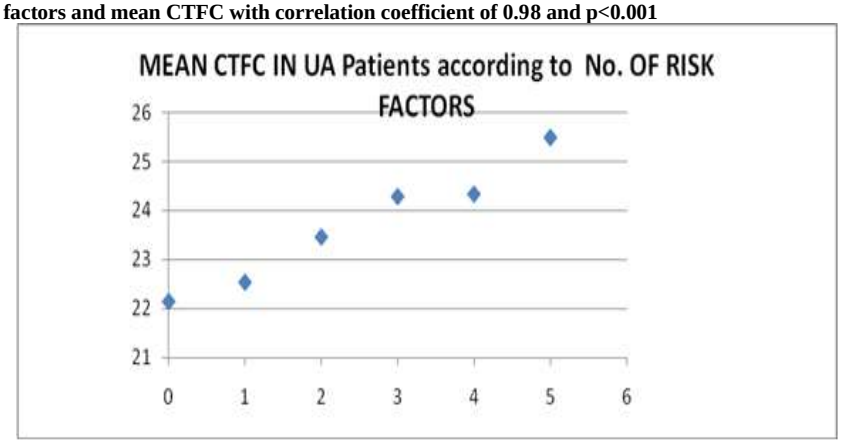
X AXIS – NUMBER OF RISK FACTORS
Y AXIS – MEAN CTFC IN CSA Patients
CORRELATION COEFFICIENT r=0.95 p VALUE OF CORRELATION p <0.001

CORRELATION BETWEEN NUMBER OF RISK FACTORS AND MEAN CTFC IN UA PATIENTS

TABLE 9 - CORRELATION BETWEEN NUMBER OF RISK FACTORS AND MEAN CTFC IN UA PATIENTS

NUMBER OF RISK FACTORS	MEAN CTFC IN UA PATIENTS
0	22.15
1	22.54
2	23.46
3	24.28
4	24.68
5	25.48

CHART 2 – Shows correlation between number of risk factors and mean CTFC in UA Patients. There was a strong correlation between no. of risk factors and mean CTFC with correlation coefficient of 0.98 and p<0.001



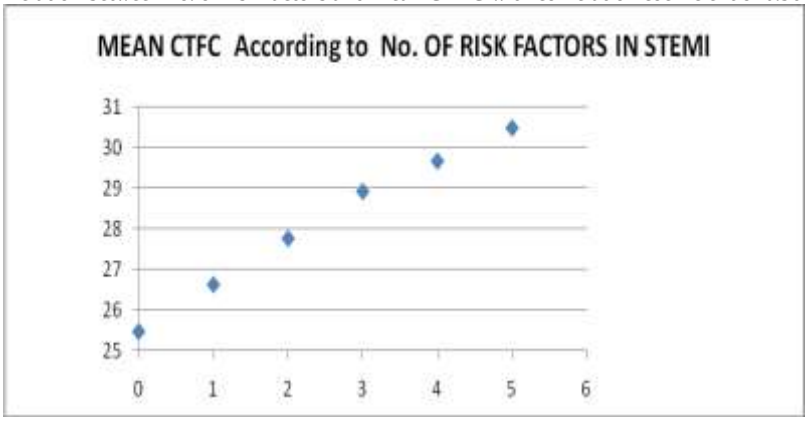
X AXIS – NUMBER OF RISK FACTORS
Y AXIS – MEAN CTFC IN CONTROLS
CORRELATION COEFFICIENT r=0.98p VALUE OF CORRELATION p <0.001

CORRELATION BETWEEN NUMBER OF RISK FACTORS AND MEAN CTFC IN STEMI PATIENTS IN NIRAS

TABLE 10 - CORRELATION BETWEEN NUMBER OF RISK FACTORS AND MEAN CTFC IN STEMI PATIENTS in NIRAS

NUMBER OF RISK FACTORS	MEAN CTFC IN STEMI PATIENTS
0	25.46
1	26.62
2	27.76
3	28.92
4	29.67
5	30.49

CHART 3 – Shows correlation between number of risk factors and mean ctfc in Non infarct related coronary arteries in NSTEMI. There was a strong correlation between no. of risk factors and mean CTFC with correlation coefficient of 0.96 and p<0.001



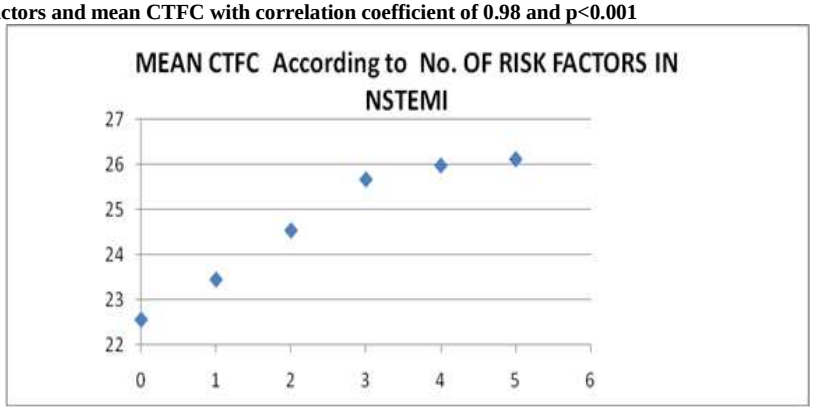
X AXIS – NUMBER OF RISK FACTORS
Y AXIS – MEAN CTFC IN CONTROLS
CORRELATION COEFFICIENT r=0.96 p VALUE OF CORRELATION p <0.001

CORRELATION BETWEEN NUMBER OF RISK FACTORS AND MEAN CTFC IN NSTEMI PATIENTS IN NONINFART RELATED ARTERIES

TABLE 11 - CORRELATION BETWEEN NUMBER OF RISK FACTORS AND MEAN CTFC IN NSTEMI PATIENTS

NUMBER OF RISK FACTORS	MEAN CTFC IN NSTEMI PATIENTS
0	22.56
1	23.45
2	24.54
3	25.67
4	25.98
5	26.12

CHART 4 – Shows correlation between number of risk factors and mean CTFC in NSTEMI. There was a strong correlation between no. of risk factors and mean CTFC with correlation coefficient of 0.98 and p<0.001



X AXIS – NUMBER OF RISK FACTORS
Y AXIS – MEAN CTFC IN CONTROLS
CORRELATION COEFFICIENT r=0.98p VALUE OF CORRELATION p <0.001

CTFC IN PATIENTS WITH DIFFERENT RISK FACTORS

TABLE 12 - CTFC IN PATIENTS WITH DIFFERENT RISK FACTORS

RISK FACTOR	MEAN CTFC				
	Overall	CSA	UA	STEMI NIRA	NSTEMI NIRA
DIABETES	26.12	22.64	24.16	29.41	25.95
HYPERTENSION	25.78	22.57	24.01	28.59	25.26
SMOKING	25.76	22.41	23.98	28.78	25.35
DYSLIPIDEMIA	26.32	22.78	24.35	29.61	25.76
FAMILY HISTORY OF PREMATURE CAD	25.58	22.31	23.80	28.35	25.15
NONE OF THE ABOVE RISK FACTORS	25.30	22.23	23.71	27.95	24.96

CHART 5 - Shows CTFC in patients with different risk factors. Dyslipidemia AND Diabetes predicted higher CTFC, followed by hypertension and smoking. However, there was no statistically significant diference

IV) CTFC AMONG DIFFERENT TYPES OF PRESENTATIONS

CTFC IN CSA PATIENTS

Mean overall CTFC – 22.53
STANDARD DEVIATION – 3.36

VESSEL WISE CTFC IN CSA PATIENTS

TABLE 13 - VESSEL WISE CTFC IN CSA PATIENTS

NAME OF CORONARY ARTERY	MEAN CTFC
LAD	22.78+3.34
RCA	22.45+3.08
LCX	22.36+3.63

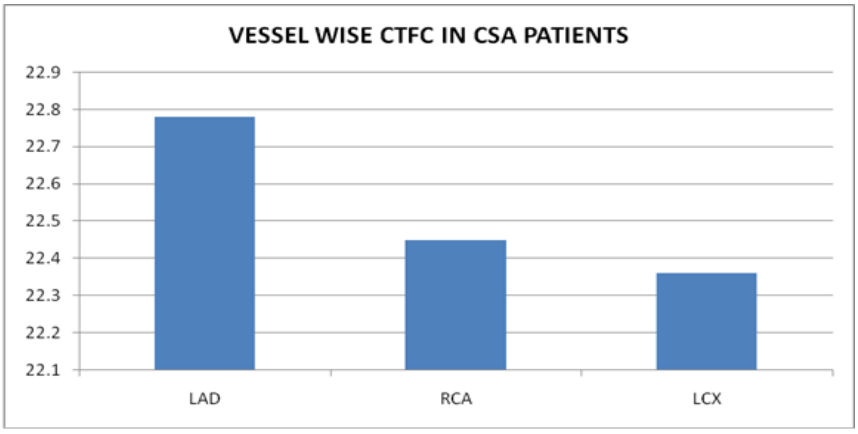


CHART 6 – Shows vessel wise CTFC in CSA for LAD, LCX and RCA separately.

CTFC IN UA PATIENTS

Mean overall CTFC – 24.41
STANDARD DEVIATION – 3.65

VESSEL WISE CTFC IN UA PATIENTS

TABLE 14- VESSEL WISE CTFC IN UA PATIENTS

NAME OF CORONARY ARTERY	MEAN CTFC
LAD	24.41+3.65
RCA	23.49+3.74
LCX	24.42+3.94

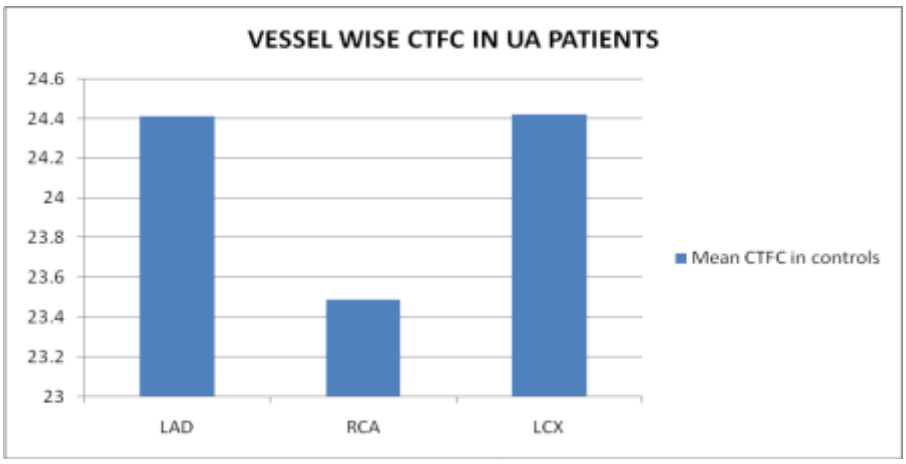


CHART 7 – Shows vessel wise CTFC in UA for LAD, LCX and RCA separately

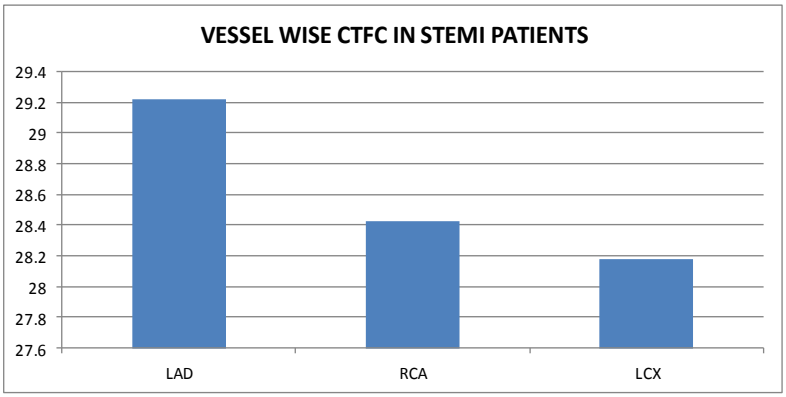
CTFC IN STEMI AND NSTEMI PATIENTS IN NON-INFARCT RELATED ARTERIES

STEMI -Mean overall CTFC in NIRAS– 28.57 STANDARD DEVIATION – 3.94
NSTEMI -Mean overall CTFC in NIRAS–25.23 STANDARD DEVIATION – 2.55

TABLE 15-VESSEL WISE CTFC IN STEMI AND NSTEMI PATIENTS IN NIRAS

NAME OF CORONARY ARTERY	MEAN CTFC	
	STEMI	NSTEMI
LAD	29.22+4.09	25.64+2.49
RCA	28.43+4.09	25.12+2.03
LCX	28.18+3.66	24.98+2.98

CHART 8-VESSEL WISE CTFC IN STEMI PATIENTS IN NIRAS



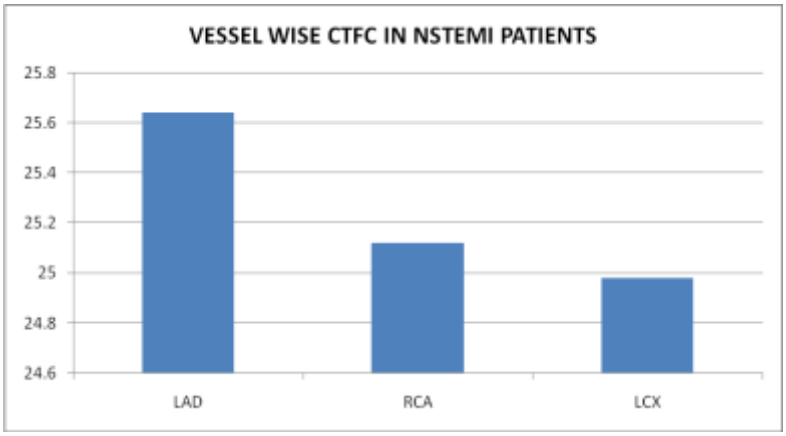


CHART9--VESSEL WISE CTFC IN NSTEMI PATIENTS IN NIRAS

CTFC IN STEMI AND NSTEMI PATIENTS IN INFARCT RELATED ARTERIES

STEMI -Mean overall CTFC in IRAS–35.63 STANDARD DEVIATION – 4.25
NSTEMI -Mean overall CTFC in IRAS– 29.48 STANDARD DEVIATION – 2.98

TABLE 16 -VESSEL WISE CTFC IN STEMI AND NSTEMI PATIENTS IN IRAS

NAME OF CORONARY ARTERY	MEAN CTFC	
	STEMI	**NSTEMI**
LAD	35.56+4.86	25.64+2.49
RCA	36.26+4.87	30.17+3.88
LCX	34.64+3.92	29.05+2.15

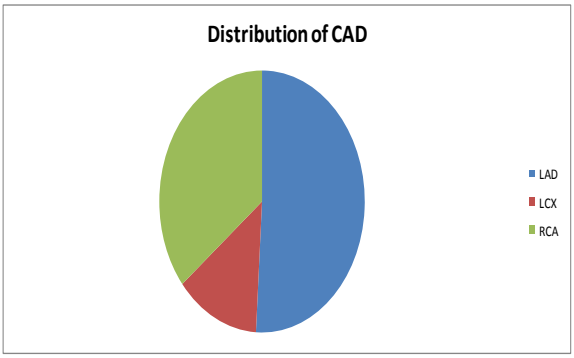
Significant slow flow was present in STEMI patients in infarct related arteries compared to NSTEMI patients. P value <0.05

LOCATION WISE DISTRIBUTION OF CAD (INFARCT RELATED ARTERY) IN STUDY SAMPLE –

TABLE 17 - LOCATION WISE DISTRIBUTION OF CAD (INFARCT RELATED ARTERY) IN STUDY SAMPLE

INFARCT RELATED ARTERY	NUMBER OF PATIENTS
LAD	108
LCX	28
RCA	76

CHART 10 – Shows location wise distribution of CAD (infarct related artery) in study sample. LAD was most commonly involved artery followed by RCA and LCX in that order.



MYOCARDIAL BLUSH GRADE

TABLE 18 - MYOCARDIAL BLUSH IN DIFFERENT PRESENTATIONS

MBG	LCX	LAD	RCA
≥2	390	365	364
0 or 1	10	35	36

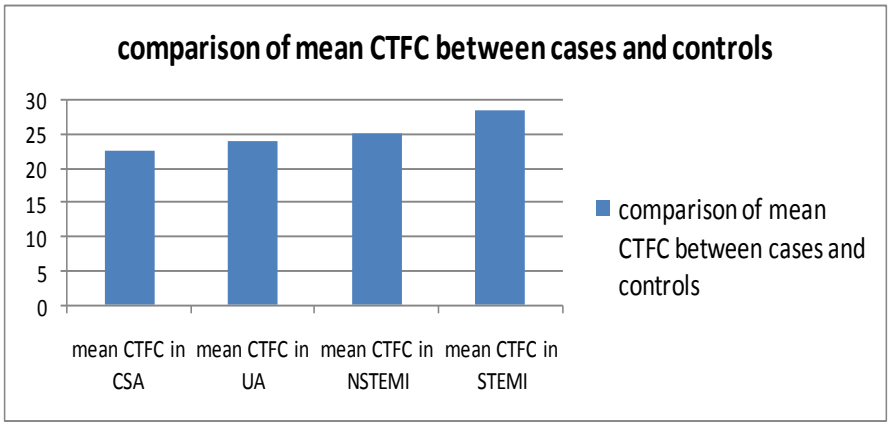
For patients with myocardial blush of 0 or 1, distribution of patients are as follows
LCX – all 10 patients presented with STEMI
LAD- 21 patients presented with STEMI, 6 patients had NSTEMI, 2 patients had UA and 6 patients had CSA
RCA- 18 patients presented with STEMI, 6 patients had NSTEMI, 4 patients had UA and 8 patients had CSA
However, myocardial blush was preserved (≥2) in all the arteries in absence of significant epicardial coronary artery stenosis.

COMPARISON OF CTFC BETWEEN DIFFERENT GROUPS

TABLE 19 - COMPARISON OF CTFC BETWEEN DIFFERENT GROUPS

STUDY GROUP	MEAN CTFC (OVERALL)
CSA	22.53, S.D = 3.36
UA	24.15, S.D. =3.77
NSTEMI non infarct related artery	25.23, S.D.=2.55
STEMI non infarct related artery	28.57, S.D.= 3.94

CHART 11 – Shows comparison of CTFC among different groups. p<0.001.



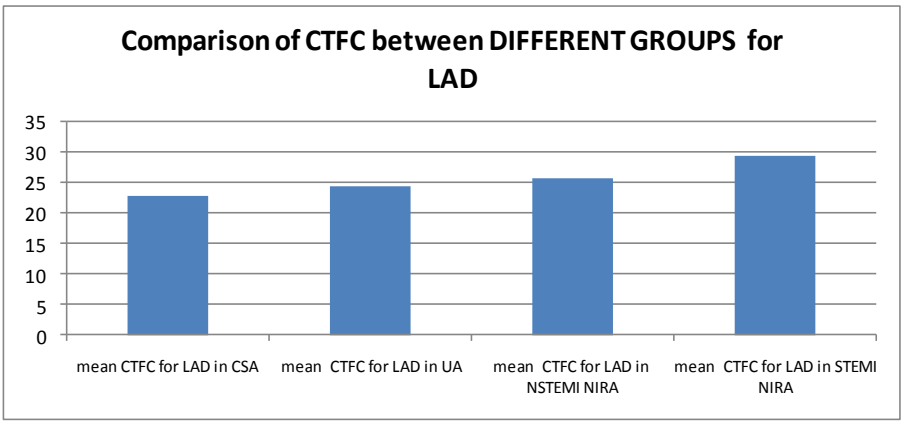
p value for significance of difference between mean CTFC among different groups, p <0.001

VESSEL WISE COMPARISON OF MEAN CTFC BETWEEN DIFFERENT GROUPS

TABLE 20 - COMPARISON OF MEAN CTFC FOR LAD BETWEEN DIFFERENT GROUPS

STUDY GROUP	MEAN CTFC FOR LAD
CSA	22.78, S.D = 3.34
UA	24.41, S.D. =3.65
NSTEMI non infarct related artery	25.64, S.D.=2.49
STEMI non infarct related artery	29.22, S.D.= 4.07

CHART 12 - Comparison of mean CTFC for LAD in DIFFERENT GROUPS

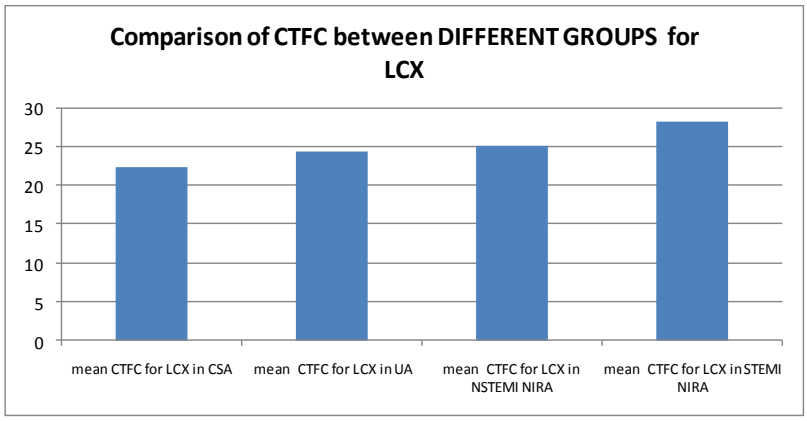


P value for significance of difference between mean CTFC in LAD for different groups, p <0.001

TABLE 21 - COMPARISON OF MEAN CTFC FOR LCX IN CASES AND CONTROLS

STUDY GROUP	MEAN CTFC FOR LAD
CSA	22.36, S.D = 3.63
UA	24.42, S.D. =2.98
NSTEMI non infarct related artery	24.98, S.D.=2.98
STEMI non infarct related artery	28.18, S.D.= 3.66

CHART 13 - Comparison of mean CTFC for LCX in different groups. P <.001

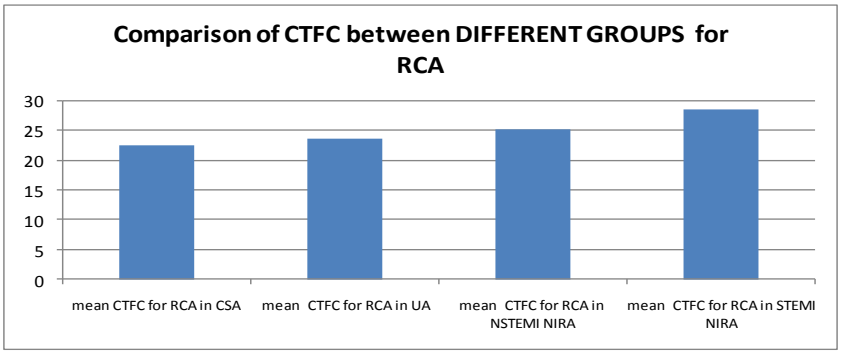


P value for significance of difference between mean CTFC for LCX in different groups - p <0.001

TABLE 22 - COMPARISON OF MEAN CTFC FOR RCA IN CASES AND CONTROLS

STUDY GROUP	MEAN CTFC FOR RCA
CSA	22.45, S.D = 3.08
UA	23.49, S.D. =3.74
NSTEMI	25.12, S.D.=2.03
STEMI	28.43, S.D.= 4.09

CHART 14 - Comparison of mean CTFC for RCA in different groups, p<0.001..



P value for significance of difference between mean CTFC for RCA in different groups- p <0.001

NORMAL CORONARY ANGIOGRAM

In our study population normal epicardial coronary arteries was present in 73 patients (18% of study population). This included patients with normal coronary arteries, those with MCVD, patients having minor plaques in coronary arteries and AMI Patients with recanalised vessels.

Normal epicardial coronary arteries were present in 37 patients with CSA, 12 patients with UA, 8 patients with NSTEMI, and 16 patients with STEMI.

Table 23- shows number and percentage of patients with normal epicardial coronary arteries with IHD

STUDY GROUP	Total No. of patients	No. of patients with normal epicardial coronary arteries	% of patients with normal epicardial coronary arteries
CSA	99	37	37.4%
UA	36	12	33.3%
NSTEMI	53	8	15.1%
STEMI	212	16	7.5%

DISCUSSION

We studied the status of coronary microcirculation in patients presenting at a tertiary care hospital with ischemic heart disease. We divided the patient population into different groups according to the presentation such as chronic stable angina, unstable angina, NSTEMI and STEMI. Corrected TIMI frame count and myocardial blush of the coronary arteries of the patients undergoing coronary angiogram was studied. Among the patient population we studied, 99 patients presented with CSA, 212 patients presented with STEMI, 36 patients presented with unstable angina and 53 presented with NSTEMI.

Male patients dominated the study population across all types of presentation. However, there was no significant difference in the mean CTFC among the male and female patients. We studied the distribution of 5 major modifiable risk factors in our study population. Of the risk factors studied, Dyslipidemia was the most common risk factor followed by smoking, hypertension, diabetes and positive family history in that order. 30% of patients had single risk factor, 36% had 2 risk factors, 16 % had 3 risk factors, 8 % had 4 risk factors and 3 % had all the risk factors. There was a strong correlation between no. of risk factors and mean CTFC with correlation coefficient of 0.98 and $p<0.001$ Dyslipidemia and diabetes predicted higher CTFC, followed by hypertension and smoking although there was no statistically significant difference. Although, the mean CTFC in diabetic patients in our study was higher than in those without any risk factors, this difference does not reach statistical significance probably because of small sample size. There was no significant difference in CTFC between smokers and non smokers

We compared mean CTFC in the patients and found that coronary flow was significantly slower in non infarct related arteries in the setting of STEMI and NSTEMI despite no obstructive epicardial coronary lesion as compared to patients presenting with chronic stable angina and unstable angina. However, myocardial blush was preserved in all the vessels (those without significant epicardial coronary artery stenosis). Among the non-infarct related arteries without any significant obstructive lesions in the STEMI and NSTEMI patients significant slow flow was present in the STEMI group as compared to NSTEMI group. Similarly significant slow flow was present in the patients with unstable angina when compared with patients presenting with stable angina. The mean CTFC in non-infarct related arteries in STEMI patients in our study was however lower than that found by Gibson CM et al who measured CTFC in NIRAs 90 minutes after thrombolysis (30.9+/- 15.0 frames[3,4]. The likely cause for this difference may be the time delay for angiography after the onset of symptoms, which in our study was 11 hours (mean). These findings suggest the

presence of microvascular dysfunction independent of epicardial event of thrombus formation or reperfusion. Comparison of CTFC in the infarct related arteries in STEMI and NSTEMI showed significant slow flow in the STEMI group.

Hypertension causes acquired hypertrophy of the left ventricle where coronary resistance vessels remain unchanged, because maximum absolute flow (ml/min) remains unchanged, maximum perfusion per gram of myocardium falls. The net effect is that coronary flow reserve at any given coronary arterial pressure is reduced and inversely related to the change in LV mass, this reduction in coronary flow reserve may result in decreased perfusion at the time of stress as in case of acute MI. Hyperlipidemia causes impaired endothelial-dependent vasodilation, particularly NO mediated vasodilation. Kuo and colleagues have demonstrated that dietary hypercholesterolemia in swine markedly attenuates the dilation of coronary arterioles in response to shear stress as well as pharmacological agonists that stimulate NO synthase in the absence of epicardial stenoses [5] This was reversed with l-arginine, suggesting that it reflects impaired NO synthesis or availability. Yokoyama I et al found that the coronary flow reserve is markedly reduced in the absence of a coronary stenosis in familial hypercholesterolemia and improving endothelial function by lowering elevated LDL levels with statins produces a delayed improvement in coronary flow reserve in normal and stenotic arteries and also ameliorates clinical signs of myocardial ischemia.

In our study population normal epicardial coronary arteries was present in 73 patients (18% of study population). This included patients with normal coronary arteries, those with MCVD, patients having minor plaques in coronary arteries and AMI Patients with recanalised vessels.

Normal epicardial coronary arteries were present in 37 patients with CSA (37.4 % of patients with CSA), 12 patients with UA (33.3% patients with UA), 8 patients with NSTEMI (15.1% patients with NSTEMI), and 16 patients with STEMI (7.1% patients with STEMI).

CONCLUSION

The presence and significance of microvascular dysfunction in ischemic heart disease is a subject of intense investigation. We studied the status of coronary microvascular function in patients with ischemic heart disease using corrected TIMI frame count and myocardial blush. We compared the values of CTFC among the various groups in our study population.

We found that coronary flow was significantly slower in non infarct related arteries in the setting of STEMI and NSTEMI despite no obstructive epicardial coronary lesion as compared to coronary flow in the absence of acute MI ($p < 0.001$). CTFC in males was more than in females but was not statistically significant ($p > 0.10$). Also, significant slow flow was present in NIRAs in STEMI as compared to NSTEMI. Similarly significant slow flow was present in the patients with unstable angina when compared with patients presenting with stable angina. The correlation between various atherosclerotic risk factors and CTFC was studied. Slower flow in NIRAs without flow limiting epicardial disease indirectly implies the presence of primary microvascular dysfunction independent of epicardial event. The amount of microvascular dysfunction was directly related to number of atherosclerotic risk factors which again suggests that microvascular dysfunction might precede epicardial thrombosis. Diabetes and dyslipidemia predicted higher CTFC.

Large studies are thus warranted in near future to develop a better understanding and thus, effective therapies directed to prevent/ treat microvascular dysfunction in the setting of IHD.

Declaration by Authors

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