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A Study on Evaluation of Diagnostic Role of NT-proBNP in Heart Failure in Background of STEMI

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Abstract: ***We have evaluated the relationship between NT-proBNP levels and symptom onset, markers of reperfusion, size of infarct and prognosis in the STEMI patients. Objectives of the study: To study diagnostic role of NT-proBNP in ST segment elevation myocardial infarction patients complicated by heart failure, clinical profile of heart failure in STEMI patients and comparison between STEMI with heart failure and STEMI without heart failure. Study Area: Department of Cardiology, Medical college, Kolkata. Study Population: Patients with STEMI admitted in Medical college Kolkata. Study Period: One year (after ethics committee clearance). Sample Size: A minimum of 80 subjects. Results: In the present study 56 males(70%) and 24 females (30%) were involved and male and female ratio was 2.33:1. 1) Present study showed that males were more commonly affected by STEMI compared to female and STEMI was also more predominant in smokers. 2) We found thatthe heart failure group of subjects had mean CPK and CPK-MB higher in comparison to that in the group without heart failure. 3) It was found that NT pro-BNP was significantly increased in heart failure group compared to group without heart failure. 4) Our study found that LVIDD and LVIDS were higher in heart failure group compared to group without heart failure which was statistically significant. Mean (+/- SD) ejection fraction was significantly lower in heart failure group. 5) Heart failure was more common in the patients with AWSTEMI compared to IWSTEMI group and it was statistically significant. 6) Heart failure was more common in the patients with triple vessel coronary artery disease and it was statistically significant. 7) All patients of AMI with and without heart failure taken together(n=80) when studied it was found that the NT pro-BNP was positively correlated with LVIDD, LVIDS and severity of CAD in STEMI patients which was statistically significant. NT pro-BNP was negatively correlated with LVEF in STEMI patient and it was statistically significant. 8) In patients of AMI with systolic heart failure group only (n=40) NT pro-BNP was positively correlated with LVIDD, LVIDS and severity of CAD but NT pro-BNP was negatively correlated with LVEF which were not statistically significant. 9) In patients of AMI with diastolic heart failure only (n=10) NT pro-BNP was positively correlated with LVIDD, LVIDS and severity of CAD. It was also observed that NT pro-BNP was negatively correlated with LVEF though that was not statistically significant. 10) It seems that the NT-proBNP in acute coronary syndrome may be a very useful marker .There is a positive correlation between NT Pro BNP and the number of coronary artery (ies) involved and the severity of luminal stenosis. Last but not the least NT Pro BNP is a very valuable marker for predicting higher incidence of heart failure and lower ejection fraction.***

Keywords: STEMI, Heart failure, NTproBNP

1. Introduction

Accurate diagnosis of heart failure is known to be a significant challenge for healthcare professionals in emergency departments (EDs). Acute myocardial infarction (AMI) is a common cause of heart failure (HF), which can develop soon after AMI and may persist or resolve or develop late. Both BNP and NT-proBNP can be detected in the circulation. Whilst increased levels of these biomarkers are not exclusive to incidences of heart failure, studies have shown that they can be sensitive and specific diagnostic biomarkers for heart failure when used as an adjunct to clinical judgment. We accordingly evaluated the relationship between NT-proBNP levels and symptom onset, markers for diagnosis of heart failure in the STEMI patients.

During the last decade, B type-natriuretic peptides have moved on »from bench to bedside« very quickly. Originally, they were introduced in clinical practice as a diagnostic tool for heart failure (HF)[1]. Later, their independent prognostic value was also shown, especially concerning mortality and heart failure, in patients with stable and unstable coronary artery disease (CAD). On the other hand, data about acute coronary events prediction are still conflicting; in contrast to the »PEACE« trial[2], in which neither BNP nor NT-proBNP significantly increased the risk of myocardial infarction (MI), »The Heart and Soul Study« found an independent association of both markers with the individual outcomes of heart failure, myocardial infarction and cardiovascular death[3]. Also, NT-proBNP was found to be a useful biomarker for distinguishing patients with long-standing hypertension who are at risk of heart failure, allowing optimization and proper treatment of these patients[4].

Patients with previous myocardial infarction represent a heterogenous group, whose prognosis differs significantly. Since traditional risk factors have less prognostic value in this secondary prevention population, they are important candidates for neurohumoral testing. Serial analyses of NT-proBNP in patients with non-ST segment elevation acute coronary syndromes (FRISC-II substudy) showed that levels measured during a chronic, relatively stable phase are a better predictor of mortality than those measured during an acute, unstable phase[5]. Also, assessment of NT-proBNP level 6 months after ST-elevation MI was a better indicator of infarct size and left ventricular function measured by cardiac magnetic resonance than baseline (admission) NT-pro BNP values[6].

Although previously thought to be equally effective for diagnostic and prognostic purposes[7], recently published data from »The Heart and Soul Study« found NT-proBNP to be

superior to BNP, when added to clinical risk factors, for net reclassification of the risk for major adverse cardiac events in patients with stable CAD.

Meta-analysis of nine prospective studies, which indicated strong association between the circulating concentration of NT-proBNP and long-term prognosis of patients with stable CAD, pointed out that although most of the included studies grouped the population according to the median or quartiles of NT-proBNP, the specific NT-proBNP, levels varied greatly among different studies, making it impossible to give a precise cut-point[8].

The diagnostic potential of natriuretic peptide concentrations in patients with acute dyspnea was described more than 10 years ago. They correlate with the invasively measured LV filling pressures.The International Collaborative for NT-proBNP Study helped defining the most appropriate cut-off values for NT-proBNP by pooling data from several single centre studies that had each suggested excellent accuracy but a wide range of optimal cut-off values (with differences in baseline characteristics including age, which was most likely responsible for this fact. We have accordingly evaluated the relationship between NT-proBNP levels and symptom onset, markers of reperfusion, size of infarct and prognosis in the STEMI patients.

Objectives of the study

1) To study diagnostic role of NT-proBNP in ST segment elevation myocardial infarction patients complicated by heart failure
2) To study clinical profile of ST segment elevation myocardial infarction (STEMI).
3) To study clinical profile of heart failure in ST segment elevation myocardial infarction
4) Comparison between STEMI with heart failure and STEMI without heart failure

2. Methodology

Study Area:
Department of Cardiology, Medical college, Kolkata

Study Population:
Patients with STEMI admitted in medical college Kolkata

Study Period:
One year (after ethics committee clearance)

Sample Size:
A minimum of 80 subjects

Sample Size Calculation:
Based on the previous study by James L. Januzzi et al.(2005) in which the exchange(rule out) cut off point was 300 pg/ml and with a sensitivity of 99%,specificity 60%, a minimum total sample size of 41 was calculated with a precision of 0.15.Thus we propsed to recruit a sample size of 80(50 diseased and 30 control).In our study we have evaluated 80 patients with STEMI of which 50 were case(with heart failure) and 30 were control(without heart failure).

Sample Design:
Consecutive eligible of both case and control

Study Design:
Cross sectional, observational, single hospital based study

Inclusion Criteria:

1) Patients with acute onset of chest pain with or without dyspnea diagnosed to have ST segment elevation myocardial infarction defined as Patients with typical chest pain for at least 20 min and positive troponin T level with ≥1 mm ST segment elevation in 2 adjacent leads (>0.2 mV in leads V1, V2, or V3), or a new left bundle branch block in ECG.(As per definition in Braunwald heart disease 11th edition).

Exclusion Criteria:

1) Patients unwilling to participate
2) Pregnant patients
3) Patient presented with cardiogenic shock or killip class 4
4) Patient with serum creatinine > 2
5) Patients who had cardiopulmonary resuscitation before admission

Method:
This is a Cross sectional, observational, single hospital based study which was include 80 selected patients with acute chest pain with or without dyspnea. The informed consent was obtained from every patient. All patients were subjected to standard 12-lead ECG immediately after admission. Patients with ST segment elevation at the J point in 2 or more consecutive leads (with the cut-off point being >0.2 mV in leads V1, V2, or V3, and >0.1 mV in the other leads) were defined as having ST elevation myocardial infarction. Among the whole study population,patients with atleast one of the following criteria will be defined as heart failure patients.

- Symptoms of CHF on admission according to Framingham criteria
- Killip class ≥2 on admission
- Killip class ≥2 at any time of hospitalization
- Left ventricular ejection fraction ≤40% at any time during hospitalization

Patients with none of these criteria were considered as patients without CHF.

50 patients of STEMI with features of heart failure was used as a case in this study, where as 30 patients of STEMI without any features of heart failure was taken as control group in this study. Transthoracic 2-dimensional echocardiography was performed within 24 h of admission. The LV end-diastolic (LVEDD) and left ventricular end-systolic diameters (LVESD) were measured according to the guidelines of the American Society of Echocardiography. The LV ejection fraction (LVEF) was calculated by the modified Simpson's method. Coronary angiography was done for determination of the culprit coronary artery (ies) or branch (es). Blood samples were taken from every patient immediately after admission for biochemical measurements of CK-MB, TnT and NT-proBNP. All analyses were performed with statistical software.

Study Tools:
Parameters under study:
1) History and clinical examination
2) Investigations:

Blood biochemistry
1) FBS/PPBS/HbA1C
2) Urea/ creatinine/Sodium,Potassium
3) Lipid profile
4) CK-MB,Troponin T,NT-proBNP
5) CBC
6) ECG
7) Chest xray
8) Echocardiography
9) Coronary angiography

Statistical Analysis Plan:
For statistical analysis data were entered into a Microsoft excel spreadsheet and then analyzed by SPSS (version 25.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables. Two-sample t-tests for a difference in mean involved independent samples or unpaired samples. Paired t-tests were a form of blocking and had greater power than unpaired tests. One-way analysis of variance (one-way ANOVA) was a technique used to compare means of three or more samples for numerical data (using the F distribution). A chi-squared test ($\chi 2$ test) was any statistical hypothesis test wherein the sampling distribution of the test statistic is a chi-squared distribution when the null hypothesis is true. Without other qualification, 'chi-squared test' often is used as short for Pearson's chi-squared test. Unpaired proportions were compared by Chi-square test or Fischer's exact test, as appropriate.

Correlation was calculated by Pearson correlation analysis. The Pearson product-moment correlation coefficient was a measure of the linear dependence between two variables X and Y. Explicit expressions that can be used to carry out various *t*-tests are given below. In each case, the formula for a test statistic that either exactly follows or closely approximates a *t*-distribution under the null hypothesis is given. Also, the appropriate degrees of freedom are given in each case. Each of these statistics can be used to carry out either a one-tailed test or a two-tailed test.

Once a *t* value is determined, a *p*-value can be found using a table of values from Student's t-distribution. If the calculated *p*-value is below the threshold chosen for statistical significance (usually the 0.10, the 0.05, or 0.01 level), then the null hypothesis is rejected in favour of the alternative hypothesis.*P*-value $\leq$ 0.05 was considered for statistically significant.

3. Results and Analysis

Table 1: Distribution of mean Age of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Age	Case	50	56.6800	9.4792	35.0000	85.0000	55.0000	0.7418
	Control	30	57.3667	8.1006	38.0000	69.0000	57.5000	

In Case, the mean age(mean± s.d.) of patients was 56.6800 ± 9.4792 and in Control, the mean age (mean± s.d.) of patients was 57.3667 ± 8.1006. The association of mean age vs two groups was not statistically significant (p=0.7418). Thus age was match in this study.

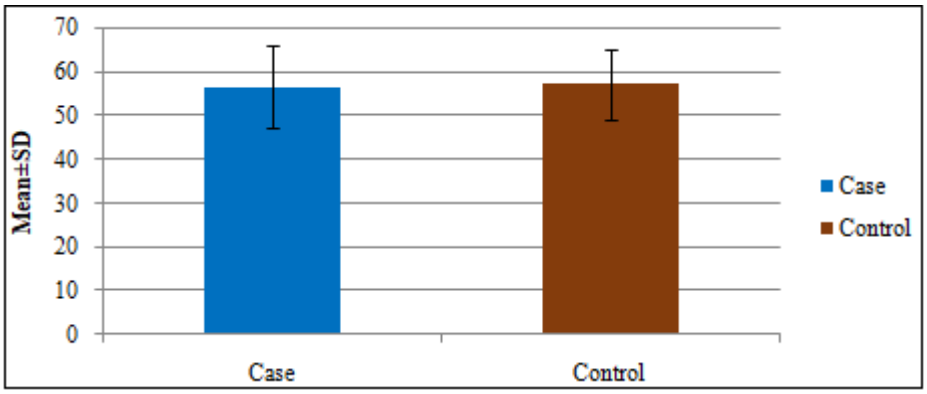

Figure 1: Distribution of mean Age of patients of STEMI

Table 2: Distribution of mean DBP and SBP of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
SBP	Case	50	131.0000	147.2220	90.0000	1146.0000	110.0000	0.8594
	Control	30	135.8000	13.0712	110.0000	164.0000	135.0000	
DBP	**Case**	50	76.4800	7.5112	62.0000	98.0000	74.0000	<0.0001
	Control	30	84.4000	8.2278	70.0000	98.0000	84.0000	

In Case, the mean SBP (mean± s.d.) of patients was 131.0000 ± 147.2220 and in control the mean SBP(mean± s.d.) of patients was 135.8000± 13.0712. The association of mean SBP vs two groups was not statistically significant (p=0.8594).

In case, the mean DBP (mean± s.d.) of patients was 76.4800 ± 7.5112 and in control, the mean DBP (mean± s.d.) of patients was 84.4000± 8.2278. The association of mean DBP vs two groups was statistically significant (p<0.0001).

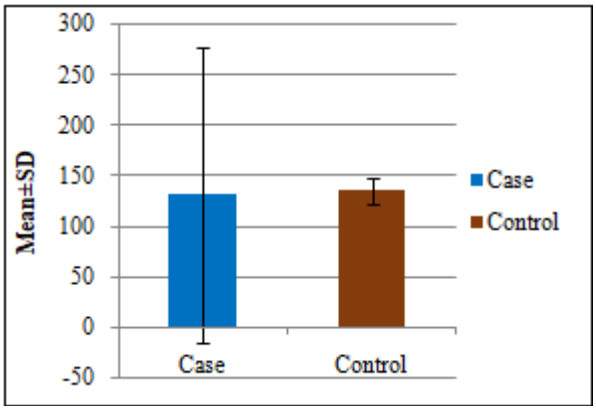

Figure 2A: Distribution of mean SBP of patients of STEMI

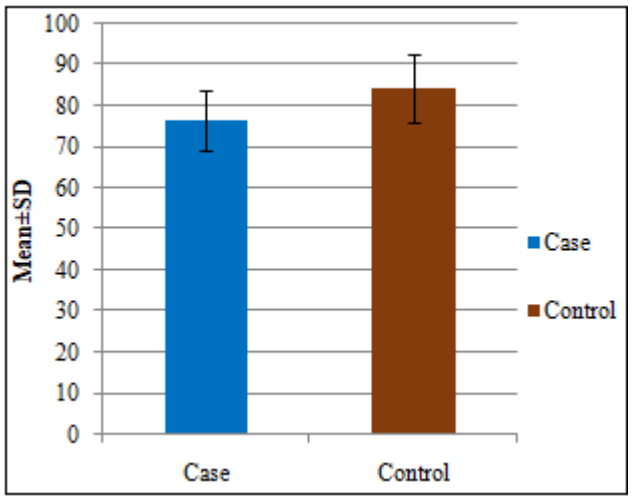

Figure 2B: Distribution of mean DBP of patients of STEMI

Table 3: Distribution of mean HR and RR of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
HR (per minute)	Case	50	100.2800	19.2301	58.0000	136.0000	102.0000	<0.0001
	Control	30	78.0667	15.3082	58.0000	120.0000	76.0000	
RR	**Case**	50	27.6200	4.2661	18.0000	36.0000	27.5000	<0.0001
	Control	30	20.2333	3.8299	14.0000	34.0000	20.0000	

In case, the mean HR (mean± s.d.) of patients was 100.2800± 19.2301(per minute)and in control, the mean HR (mean± s.d.) of patients was 78.0667± 15.3082(per minute). The association of mean HR vs two groups was statistically significant (p<0.0001).

In case, the mean RR (mean± s.d.) of patients was 27.6200± 4.2661and in control, the mean RR (mean± s.d.) of patients was 20.2333± 3.8299. The association of mean RR vs two groups was statistically significant (p<0.0001).

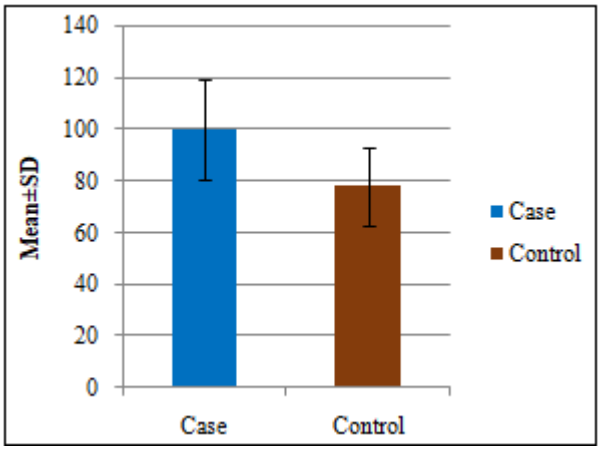

Figure 3A: Distribution of mean HR of patients of STEMI

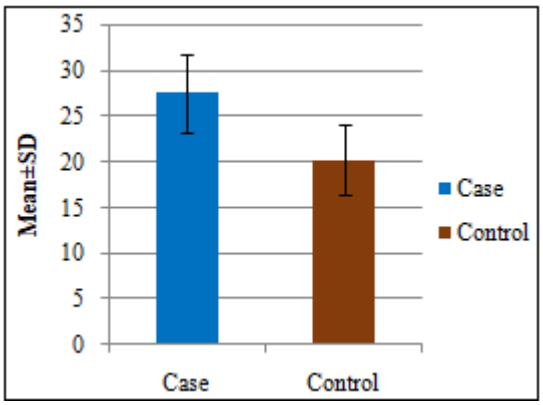

Figure 3B: Distribution of mean RR of patients of STEMI

Table 4: Distribution of mean HB of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Hb(g/dl)	Case	50	12.8140	1.3163	9.9000	16.0000	12.7500	0.9813
	Control	30	12.8067	1.4083	10.4000	15.2000	12.4500	

In case, the mean Hb(mean± s.d.) of patients was 12.8140± 1.3163g/dl and in control, the mean Hb (mean± s.d.) of patients was 12.8067± 1.4083g/dl. The association of mean Hb vs two groups was not statistically significant (p=0.9813).

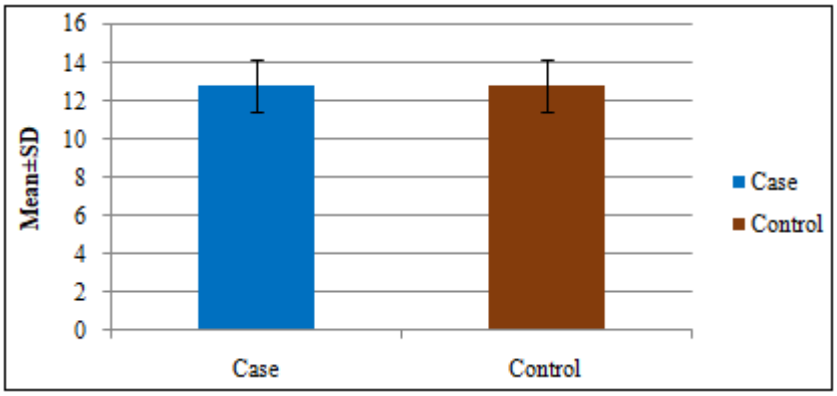

Figure 4: Distribution of mean HB of patients of STEMI

Table 5: Distribution of mean TLC of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
TLC(Cells per liter)	Case	50	9745.5200	3194.0871	4769.0000	17651.0000	9113.0000	0.3644
	Control	30	10426.4000	3293.1861	5432.0000	18761.0000	11110.5000	

In case, the mean TLC (mean± s.d.) of patients was 9745.5200± 3194.0871(Cells per liter) and in control, the mean TLC (mean± s.d.) of patients was 10426.4000± 3293.1861(Cells per liter). The association of mean TLC vs two groups was not statistically significant (p=0.3644).

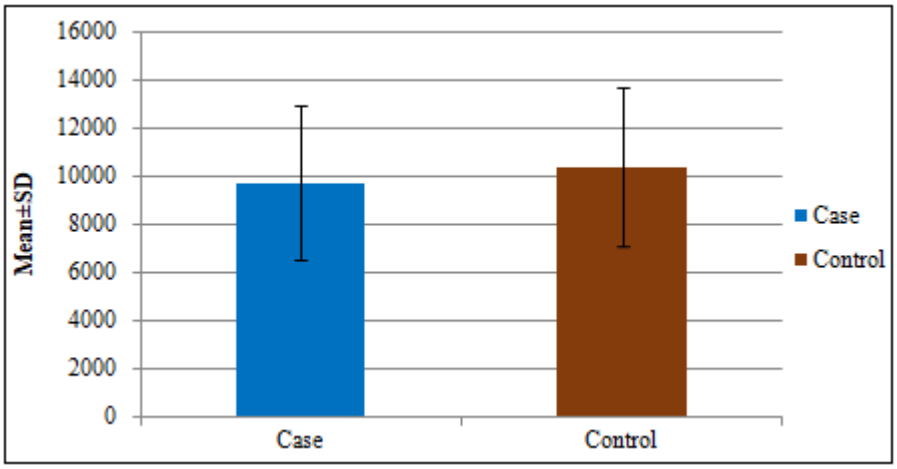

Figure 5: Distribution of mean TLC of patients of STEMI

Table 6: Distribution of mean Urea and Creatinine: Group

		Number	**Mean**	**SD**	**Minimum**	**Maximum**	**Median**	**p-value**
Urea(mg/dl)	Case	50	24.6000	10.4256	11.0000	51.0000	23.0000	0.0429
	Control	30	29.5333	10.3048	12.0000	51.0000	31.5000	
Creatinine (mg/dl)	**Case**	50	1.0280	0.1980	0.7000	1.5000	1.0000	0.4026
	Control	30	1.0667	0.2006	0.8000	1.7000	1.0000	

In case, the mean Urea (mean± s.d.) of patients was 24.6000± 10.4256(mg/dl)and in control, the mean Urea (mean± s.d.) of patients was 29.5333± 10.3048 (mg/dl). The association of mean Urea vs two groups was statistically significant (p=0.0429). In case, the mean Creatinine (mean± s.d.) of patients was 1.0280± 0.1980(mg/dl) and in control, the mean Creatinine (mean± s.d.) of patients was 1.0667± 0.2006 (mg/dl). The association of mean Creatinine vs two groups was not statistically significant (p=0.4026).

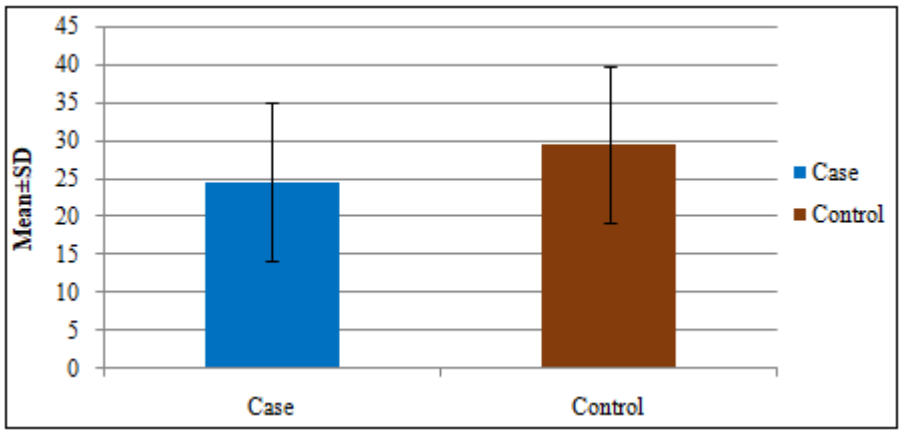

Figure 6A: Distribution of mean Urea of patients of STEMI

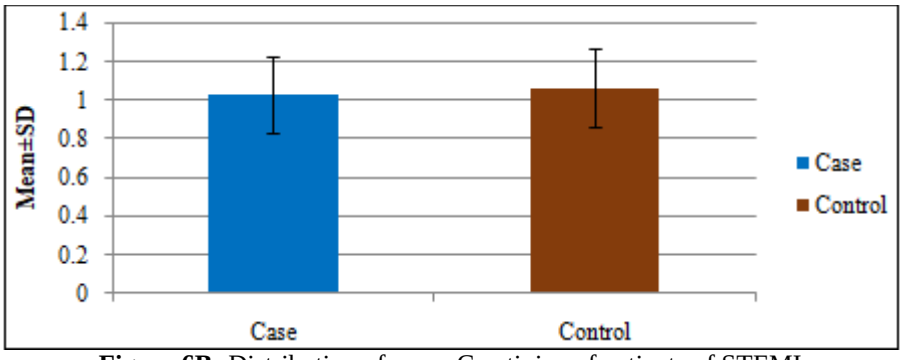

Figure 6B: Distribution of mean Creatinine of patients of STEMI

Table 7: Distribution of mean Serum Na and Serum K: Group

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Serum Na	Case	50	138.5400	5.0354	123.0000	151.0000	138.0000	0.9755
	Control	30	138.5000	6.4954	121.0000	151.0000	138.0000	
Serum K	**Case**	50	4.4520	0.6649	3.3000	5.7000	4.4000	0.8296
	Control	30	4.4833	0.5608	3.3000	5.6000	4.4000	

In case, the mean Serum Na(mean± s.d.) of patients was 138.5400± 5.0354and in control, the mean Serum Na (mean± s.d.) of patients was 138.5000± 6.4954. The association of mean Serum Na vs two groups was not statistically significant (p=0.9755).

In case, the mean Serum K(mean± s.d.) of patients was 4.4520± 0.6649and in control, the mean Serum K (mean± s.d.) of patients was 4.4833± 0.5608. The association of mean Serum K vs two groups was not statistically significant (p=0.8296).

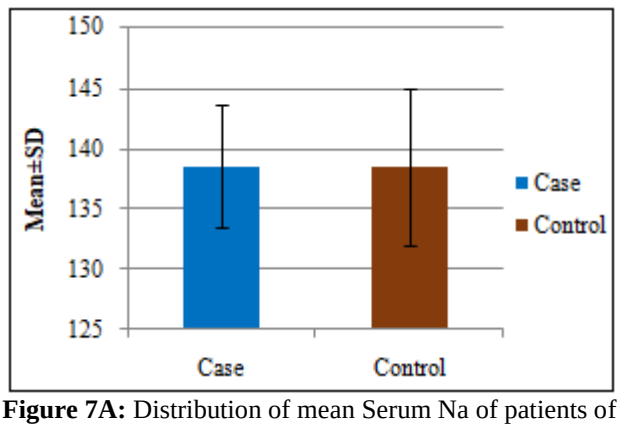

Figure 7A: Distribution of mean Serum Na of patients of STEMI

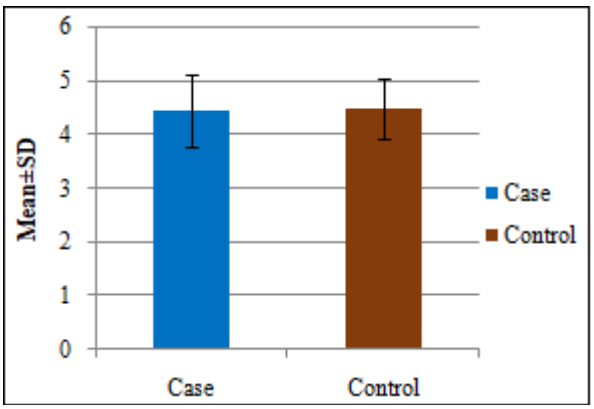

Figure 7B: Distribution of mean Serum K of patients of STEMI

Table 8: Distribution of mean CPK and CPK-MB of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
CPK	Case	50	1570.9200	1582.8160	79.0000	6543.0000	1107.0000	0.7831
	Control	30	1473.2000	1440.5551	107.0000	5432.0000	722.0000	
CPK-MB	**Case**	50	179.7600	241.2701	12.0000	1544.0000	99.5000	0.2651
	Control	30	126.8667	116.6326	10.0000	367.0000	72.5000	

In case, the mean CPK (mean± s.d.) of patients was 1570.9200± 1582.8160and in control, the mean CPK (mean± s.d.) of patients was 1473.2000± 1440.5551. The association of mean CPKvs two groups was not statistically significant (p=0.7831).

In case, the mean CPK-MB (mean± s.d.) of patients was 179.7600± 241.2701and in control, the mean CPK-MB (mean± s.d.) of patients was 126.8667± 116.6326. The association of mean CPK-MB vs two groups was not statistically significant (p=0.2651).

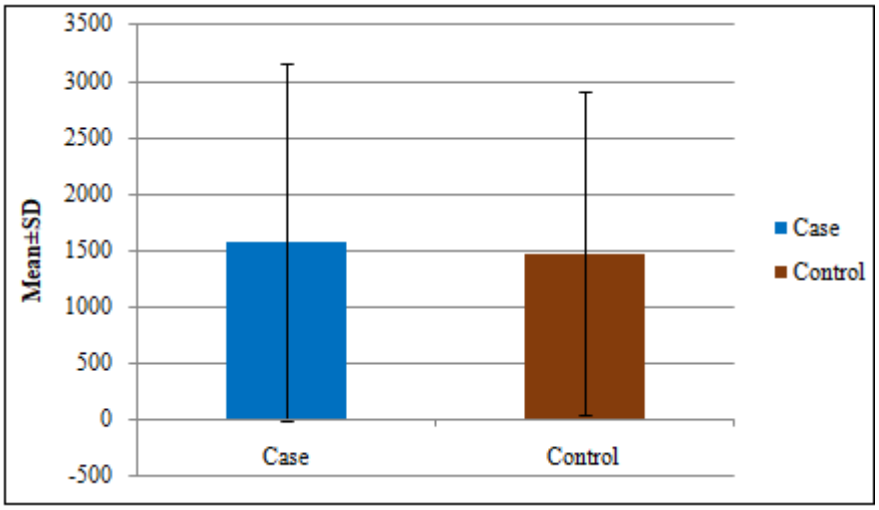

Figure 8A: Distribution of mean CPK of patients of STEMI

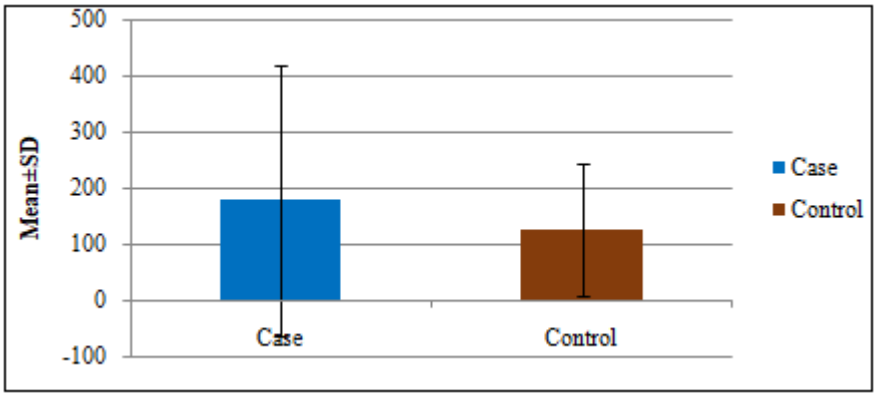

Figure 8B: Distribution of mean CPK-MB of patients of STEMI

Table 9: Distribution of mean NT pro-BNP of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
NT pro-BNP (pg/mL)	Case	50	4206.2600	3985.7472	598.0000	15257.0000	2401.0000	<0.0001
	Control	30	382.8333	176.8588	104.0000	745.0000	378.5000	

In case, the mean NT pro-BNP(mean± s.d.) of patients was 4206.2600± 3985.7472(pg/mL) and in control, the mean NT pro-BNP (mean± s.d.) of patients was 382.8333± 176.8588(pg/mL). The association of mean NT pro-BNP vs two groups was statistically significant (p<0.0001).

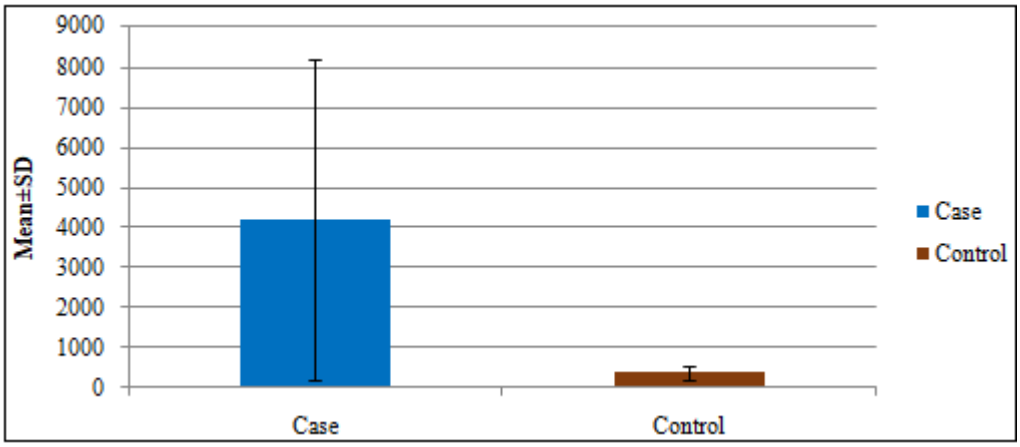

Figure 9: Distribution of mean NT pro-BNP of patients of STEMI

Table 10: Distribution of mean LVIDD of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
LVIDD	Case	50	51.2000	4.2952	43.0000	59.0000	51.0000	<0.0001
	Control	30	44.4000	2.0103	41.0000	48.0000	45.0000	

In case, the mean LVIDD(mean± s.d.) of patients was 51.2000± 4.2952and in Control, the mean LVIDD(mean± s.d.) of patients was 44.4000± 2.0103. The association of mean LVIDDvs two groups was statistically significant (p<0.0001).

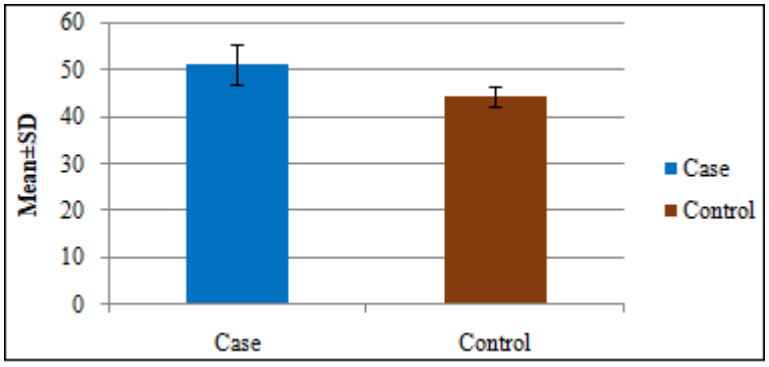

Figure 10: Distribution of mean LVIDD of patients of STEMI

Table 11: Distribution of mean LVIDS of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
LVIDS	Case	50	38.5400	5.1079	29.0000	47.0000	40.0000	<0.0001
	Control	30	30.3667	2.0424	27.0000	35.0000	30.5000	

In case, the mean LVIDD(mean± s.d.) of patients was 38.5400± 5.1079and in Control, the mean LVIDD (mean± s.d.) of patients was 30.3667± 2.0424. The association of mean LVIDSvs two groups was statistically significant (p<0.0001).

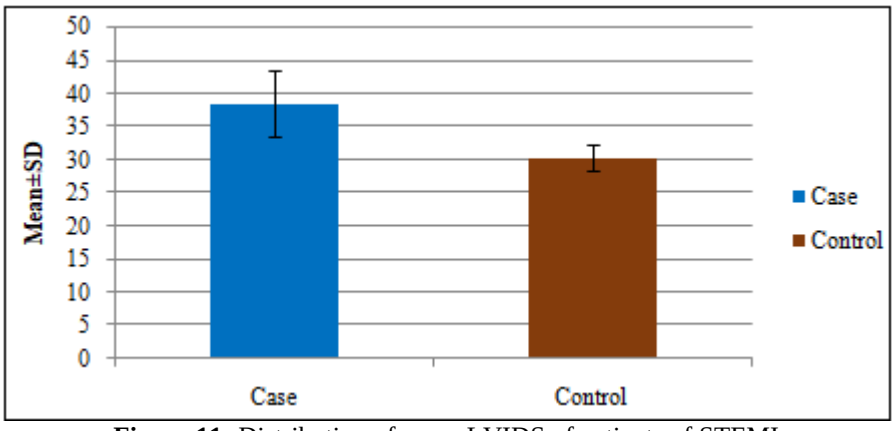

Figure 11: Distribution of mean LVIDS of patients of STEMI

Table 12: Distribution of mean LVEF of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value

LVEF	Case	50	36.6200	4.4762	30.0000	47.0000	36.5000	<0.0001
	Control	30	46.6000	2.4719	41.0000	52.0000	47.0000	

In case, the mean LVEF (mean± s.d.) of patients was 36.6200± 4.4762and in Control, the mean LVEF (mean± s.d.) of patients was 46.6000± 2.4719. The association of mean between LVEFvs two groups was statistically significant (p<0.0001).

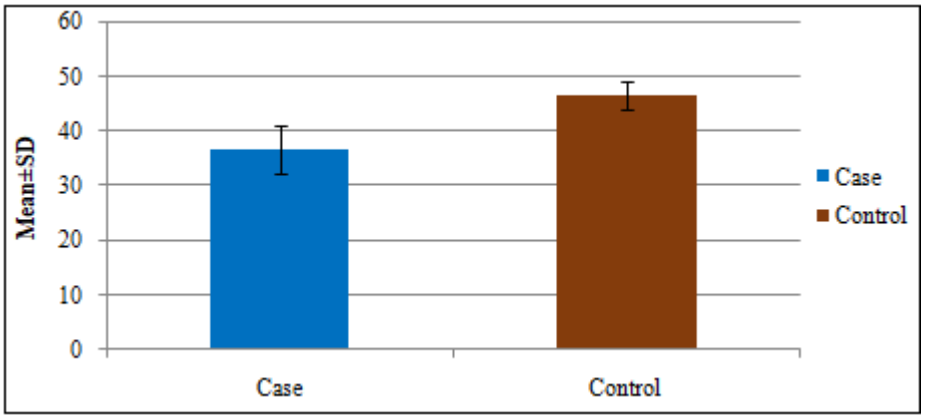

Figure 12: Distribution of mean LVEF of patients of STEMI

Table 13: Distribution of mean Grade of Diastolic dysfunction of patients of STEMI

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Grade of Diastolic dysfunction	Case	50	0.8600	0.9260	0.0000	3.0000	1.0000	<0.0001
	Control	30	0.0000	0.0000	0.0000	0.0000	0.0000	

In case, the mean Grade of Diastolic dysfunction (mean± s.d.) of patients was 0.8600± 0.9260 which was statistically significant (p<0.0001)

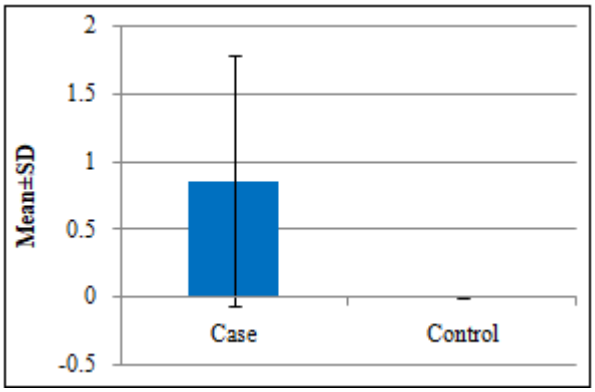

Figure 13: Distribution of mean Grade of Diastolic dysfunction of patients of STEMI

Table 14: Distribution of Occupational profile of patients of STEMI

GROUP			
Occupation	Case	Control	Total
Carpenter	1	0	1
Row %	100	0	100
Col %	2	0	1.3
Driver	2	0	2
Row %	100	0	100
Col %	4	0	2.5
Factory worker	2	1	3
Row %	66.7	33.3	100
Col %	4	3.3	3.8
Farmer	13	2	15
Row %	86.7	13.3	100
Col %	26	6.7	18.8
Fruit seller	0	2	2
Row %	0	100	100
Col %	0	6.7	2.5
Housewife	12	11	23
Row %	52.2	47.8	100
Col %	24	36.7	28.8
Juice maker	1	0	1
Row %	100	0	100
Col %	2	0	1.3
Labour	7	8	15
Row %	46.7	53.3	100
Col %	14	26.7	18.8
Office clerk	3	2	5
Row %	60	40	100
Col %	6	6.7	6.3
Security Worker	2	2	4
Row %	50	50	100
Col %	4	6.7	5
Teacher	5	1	6
Row %	83.3	16.7	100
Col %	10	3.3	7.5
Traffic surgeon	2	1	3
Row %	66.7	33.3	100
Col %	4	3.3	3.8
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 13.5575; **p-value:** 0.2585

In case, 13(26.0%) patients were Farmer and in control, 2(6.7%) patients were farmer.

In case, 12(24.0%) patients were Housewife and in control, 11(36.7%) patients were Housewife.

The association between Occupation vs two groups was not statistically significant (p=0.2585).

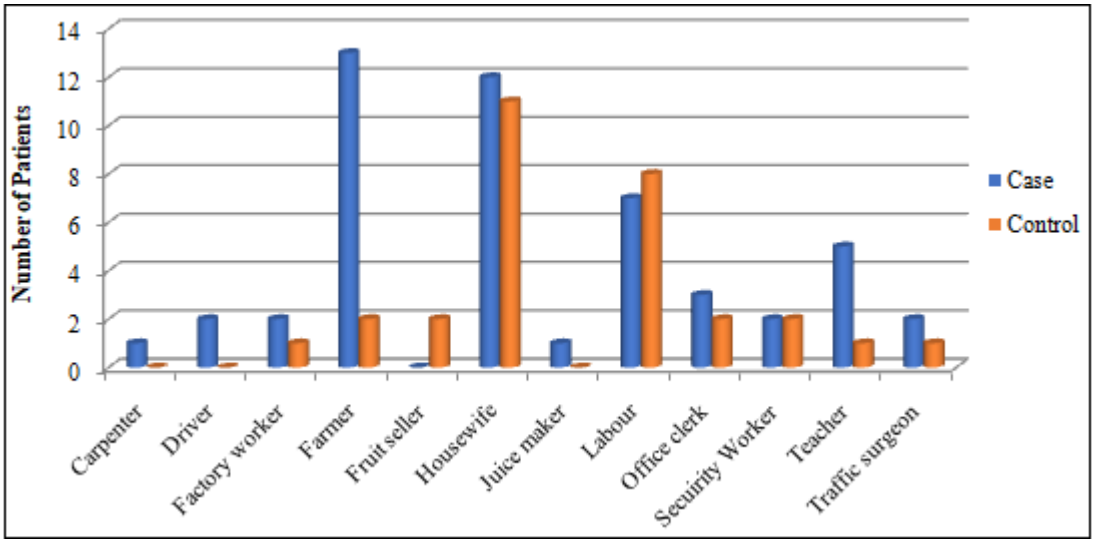

Figure 14: Distribution of Occupational profile of patients of STEMI

Table 15: Distribution of Sex of patients of STEMI

	GROUP		
Sex	Case	Control	TOTAL
Female	13	11	24
Row %	54.2	45.8	100
Col %	26	36.7	30
Male	37	19	56
Row %	66.1	33.9	100
Col %	74	63.3	70
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 1.0159; **p-value:** 0.3135

In case, 13(26.0%) patients were female and in control, 37(74.0%) patients were male. Incontrol,11(36.7%) patients were female and 19(63.3%) patients were male. The association between sex vs two groups was not statistically significant (p=0.3135).

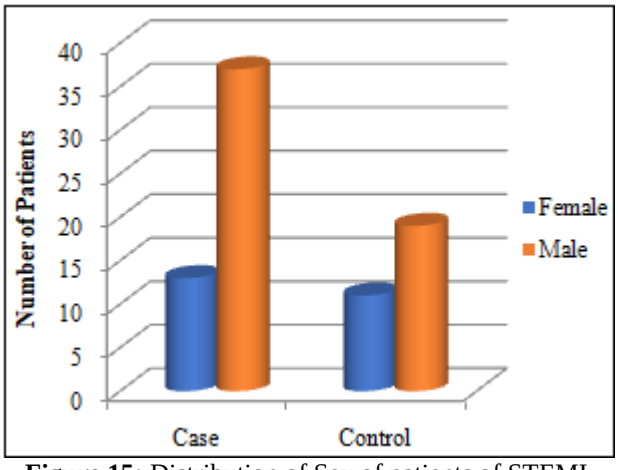

Figure 15: Distribution of Sex of patients of STEMI

Table 16: Distribution of HTN of patients of STEMI

	GROUP		
HTN	Case	Control	TOTAL
No	23	15	38
Row %	60.5	39.5	100
Col %	46	50	47.5
Yes	27	15	42
Row %	64.3	35.7	100
Col %	54	50	52.5
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 0.1203; **p-value:**0.7287

In case, 27(54.0%) patients had HTN and in control, 15(50.0%) patients had HTN. The association between HTN vs two groups was not statistically significant (p=0.7287)

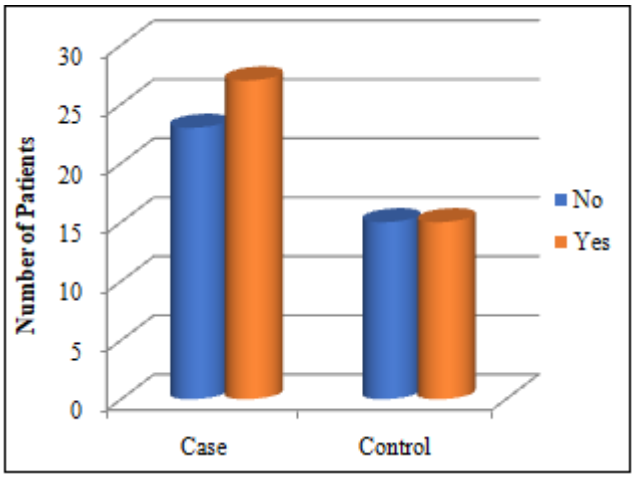

Figure 16: Distribution of HTN of patients of STEMI

Table 17: Distribution of DM of patients of STEMI

	GROUP		
DM	Case	Control	Total
No	26	11	37
Row %	70.3	29.7	100
Col %	52	36.7	46.3
Yes	24	19	43
Row %	55.8	44.2	100
Col %	48	63.3	53.8
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 1.7733; **p-value:** 0.1829

In case, 24(48.0%) patients had DM and in control, 19(63.3%) patients had DM. This association between DM vs two groups was not statistically significant (p=0.1829).

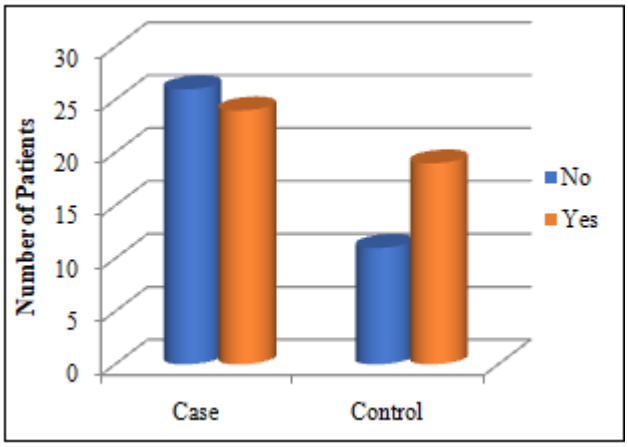

Figure 17: Distribution of DM of patients of STEMI

Table 18: Distribution smoking of patients of STEMI

	Group		
Smoker	Case	Control	Total
No	20	14	34
Row %	58.8	41.2	100
Col %	40	46.7	42.5
Yes	30	16	46
Row %	65.2	34.8	100
Col %	60	53.3	57.5
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 0.3410; **p-value:** 0.5592

In case, 30(60.0%) patients were Smoker and in control, 16(53.3%) patients were smoker. The association between Smokervs two groups was not statistically significant (p=0.5592)

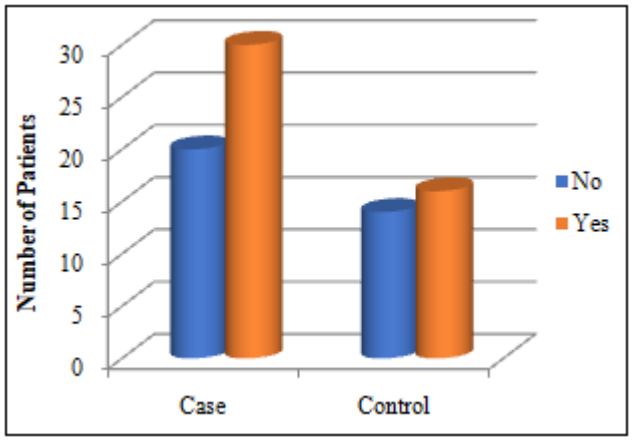

Figure 18: Distribution of smoking of patients of STEMI

Table 19: Distribution COPD of patients of STEMI

	GROUP		
COPD	**Case**	**Control**	**TOTAL**
No	40	25	65
Row %	61.5	38.5	100
Col %	80	83.3	81.3
Yes	10	5	15
Row %	66.7	33.3	100
Col %	20	16.7	18.8
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 0.1368; **p-value:** 0.7115

In case, 10(20.0%) patients had COPD and in control, 5(16.7%) patients had COPD. The association between COPD vs two groups was not statistically significant (p=0.7115).

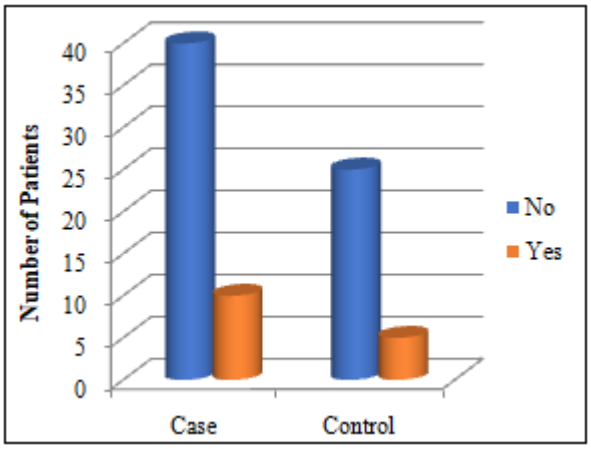

Figure 19: Distribution COPD of patients of STEMI

Table 20: Distribution of Clinical profile of patients of STEMI (N=80)

Clinical profile	Case		Total	Control		Total
	Yes	No		Yes	No	
Dyspnea	43	7	50	2	28	30
Orthopnea	31	19	50	0	30	30
PND	24	26	50	1	29	30
Fatigue	25	25	50	13	17	30
Lung Rales	26	24	50	5	25	30
JVP	24	26	50	0	30	30
S3	23	27	50	5	25	30

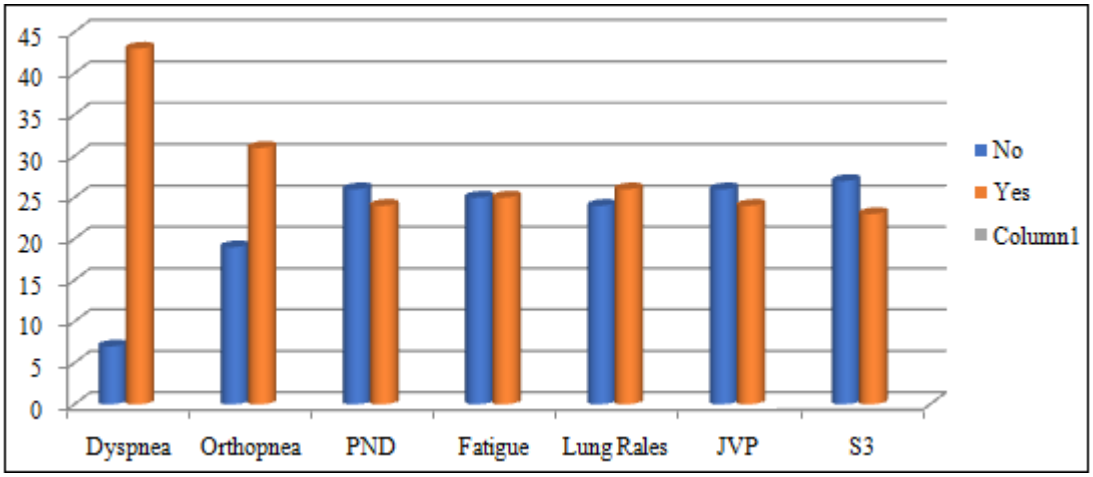

Figure 20 A: Distribution of Clinical profile of patients of STEMI with heart failure (N=50)

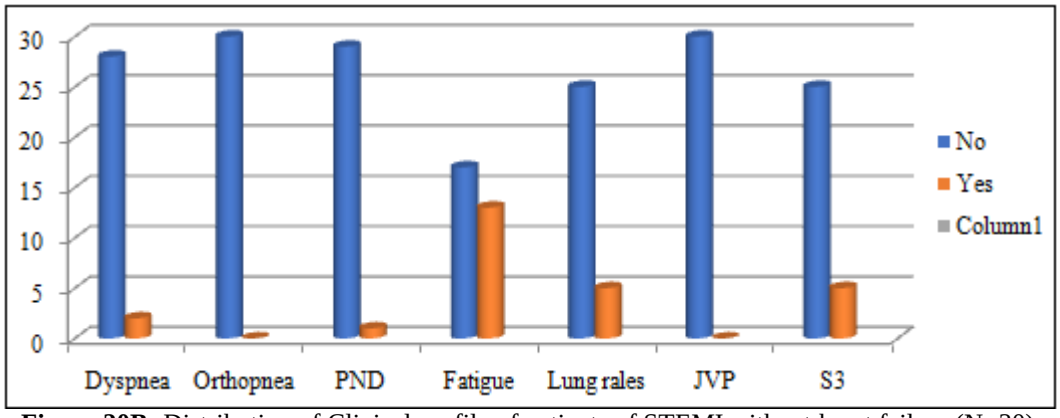

Figure 20B: Distribution of Clinical profile of patients of STEMI without heart failure (N=30)

In case, 43(86.0%) patients had Dyspnea and in control, 2(6.7%) patients had Dyspnea. The association between Dyspnea vs two groups was statistically significant (p <0.0001).

In case, 31(62.0%) patients had Orthopne and in control, no patients had Orthopnea. The association between Orthopnea vs two groups was statistically significant (p <0.0001).

In case, 24(48.0%) patients had PND and in control, 1(3.3%) patient had PND. The association between PND vs two groups was statistically significant (p <0.0001).

In case, 25(50.0%) patients had Fatigue and in control, 13(43.3%) patients had Fatigue. The association between Fatigue vs two groups was not statistically significant (p =0.5632).

In case, 26(52.0%) patients had Lung Rales and in control, 5(16.7%) patients had Lung Rales. The association between Lung Rales vs two groups was statistically significant (p=0.0016).

In case, 26(52.0%) patients had JVP NR and in control, 30(100.0%) patients had JVP NR.

In case, 24(48.0%) patients had JVP R and in control, no patients had JVP R.

The association between JVP vs two groups was statistically significant (p<0.0001).

In case, 23 (46.0%) patients had S3 and in control, 5(16.7%) patients had S3. The association between S3 vs two groups was statistically significant (p=0.0077).

Table 21: Distribution of ECG characteristics of patients of AMI (n=80)

	GROUP		
ECG	Case	Control	Total
AWSTEMI	41	7	48
IWSTEMI	9	23	32
Total	50	30	80

Chi-square value: 26.8889; **p-value:**<0.0001

In case, 41(82.0%) patients had ECG for AWSTEMI and in control, 7(23.3%) patients had ECG for AWSTEMI.

In case, 9(18.0%) patients had ECG for IWSTEMI and in control, 23(76.7%) patients had ECG for IWSTEMI.

The association between ECG vs two groups was statistically significant (p<0.0001).

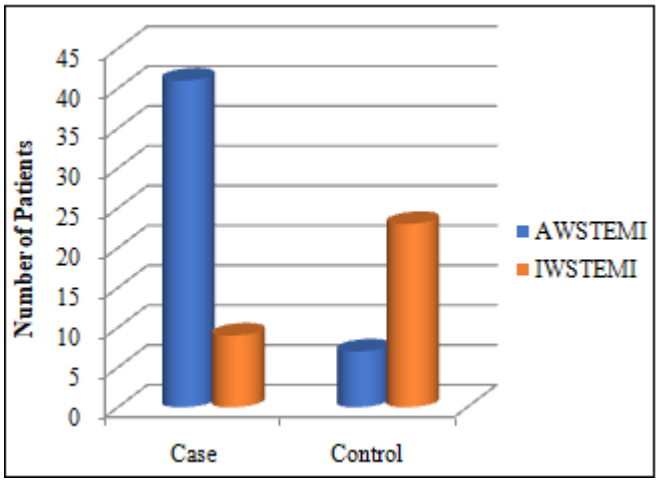

Figure 21: Distribution of ECG characteristics of patients of AMI

Table 22: Distribution of Echocardiographic RWMA characteristics in patients of STEMI (N=80)

GROUP			
RWMA	Case	Control	Total
Anterior and antero-lateral wall hypokinesia	6	1	7
Row %	85.7	14.3	100
Col %	12	3.3	8.8
Anterior and antero-septal wall hypokinesia from base to apex	27	0	27
Row %	100	0	100
Col %	54	0	33.8
Apical hypokinesia	0	2	2
Row %	0	100	100
Col %	0	6.7	2.5
Mid-apical anterior and antero-septal wall hypokinesia	9	4	13
Row %	69.2	30.8	100
Col %	18	13.3	16.3
Mid-basal inferior and infero-septal wall hypokinesia	8	23	31
Row %	25.8	74.2	100
Col %	16	76.7	38.8
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 39.2027; **p-value:** <0.0001

In case, RWMA was higher [27(54.0%)] in anterior and antero-septal wall hypokinesia from base to apex and in control, RWMA was higher [23(76.7%)] in mid-basal inferior and infero-septal wall hypokinesia. The association between RWMA vs two groups was statistically significant (p<0.0001).

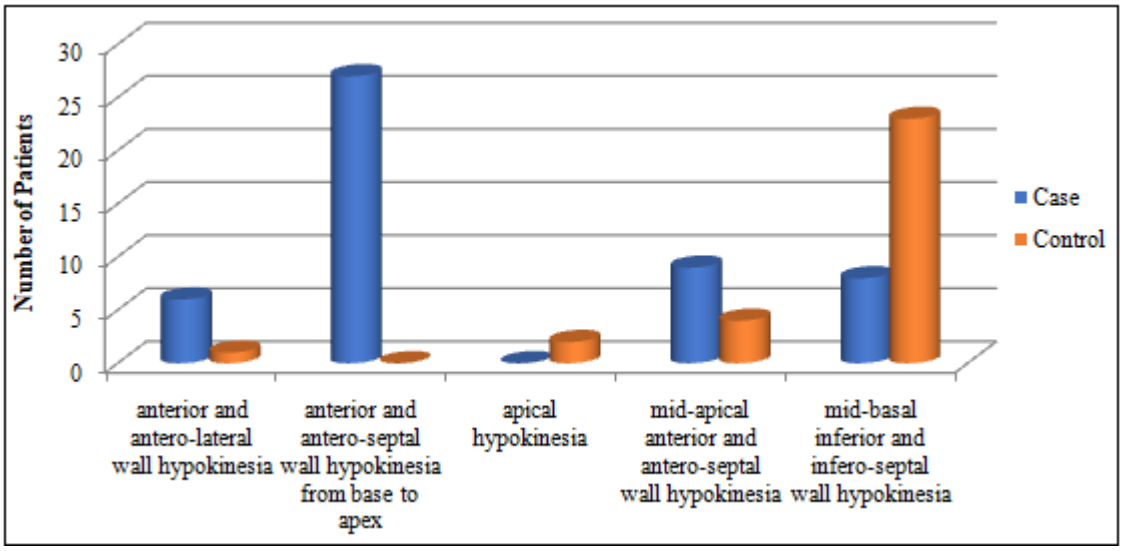

Figure 22: Distribution of Echocardiographic RWMA characteristics in patients of STEMI

Table 23: Grades of Diastolic dysfunction of patients of STEMI

GROUP			
Grade of Diastolic dysfunction	Case	Control	Total
0	23	30	53
Row %	43.4	56.6	100
Col %	46	100	66.3
1	13	0	13
Row %	100	0	100
Col %	26	0	16.3
2	12	0	12
Row %	100	0	100
Col %	24	0	15
3	2	0	2
Row %	100	0	100
Col %	4	0	2.5
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 24.4528; **p-value:** <0.0001

In case, Grade of Diastolic dysfunction was higher [23(46.0%)] in ZERO and in control, Grade of Diastolic dysfunction was higher [30(100.0%)] in ZERO. The association between Grade of Diastolic dysfunction vs two groups was statistically significant (p<0.0001).

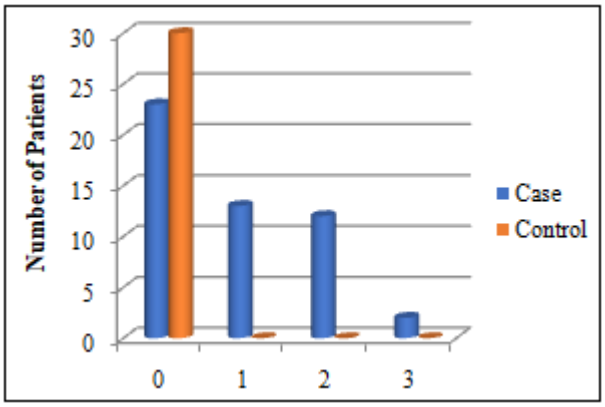

Figure 23: Grades of Diastolic dysfunction of patients of STEMI

Table 24: Distribution of number of coronary artery involved of patients of STEMI

	Group		
CAG Report	Case	Control	Total
DVCAD	23	14	37
Row %	62.2	37.8	100
Col %	46	46.7	46.3
SVCAD	13	15	28
Row %	46.4	53.6	100
Col %	26	50	35
TVCAD	14	1	15
Row %	93.3	6.7	100
Col %	28	3.3	18.8
TOTAL	50	30	80
Row %	62.5	37.5	100
Col %	100	100	100

Chi-square value: 9.1720; **p-value:** 0.0102

In case, CAG Report was higher [23(46.0%%)] in DVCAD and in control, CAG Report was higher [15(50.0%)] in SVCAD. The association between CAG Report vs two groups was statistically significant (p=0.0102).

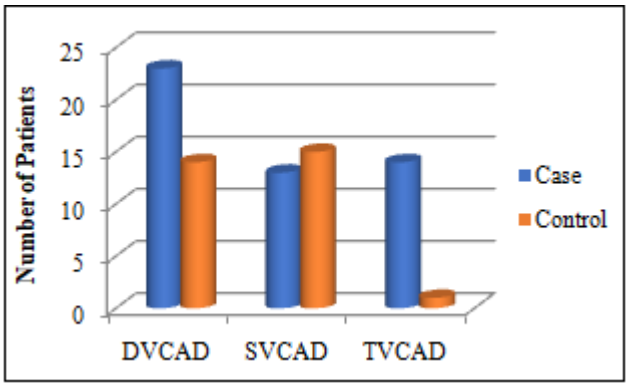

Figure 24: Distribution of number of coronary artery involved of patients of STEMI

Table 25: Mean and SD of NT Pro BNP Levels in different subsets of STEMI Patients

Mean and SD of NT Pro BNP Levels in different subsets of STEMI Patients			
	Mean	Std. Deviation	N
Total number of Stemi patients with and without heart failure			
NT pro-BNP	2772.48	3651.649	80
LVIDD	48.65	4.889	80
LVIDS	35.48	5.794	80
LVEF	40.36	6.190	80
Number of vessel involved	1.84	.719	80
TOTAL number of stemi patients with heart failure			
NT pro-BNP	4206.26	3985.747	50
LVIDD	51.20	4.295	50
LVEF	36.62	4.476	50
LVIDS	38.54	5.108	50
Number of vessel involved	2.02	.742	50

Mean and SD of NT Pro BNP Levels in different subsets of AMI Patients			
	Mean	Std. Deviation	N
STEMI WITHOUT HEART FAILURE			
NT pro-BNP	382.83	176.859	30
LVIDD	44.40	2.010	30
LVEF	46.60	2.472	30
LVIDS	30.37	2.042	30
Number of vessel involved	1.53	.571	30
STEMI WITH SYSTOLIC HEART FAILURE			
NT pro-BNP	4812.93	4225.728	40
LVIDD	52.45	3.644	40
LVEF	35.00	3.289	40
LVIDS	40.05	4.326	40
Number of vessel involved	2.15	.736	40
STEMI WITH DIASTOLIC HEART FAILURE			
NT pro-BNP	1779.60	966.099	10
LVIDD	46.20	2.860	10
LVEF	43.10	1.969	10
LVIDS	32.50	3.206	10
Number of vessel involved	1.50	.527	10

Table 26: Correlation and comparison between independent variables and NT Pro BNP (N=80)

Independent Variables	r and p values (Correlation coefficient and significance)	Dependent(out put) Variable NT pro-BNP	Remarks
LVIDD	Pearson Correlation Coefficient (r)	.522**	Positive correlation
	p-Value	<0.0001	Significant
	Number	80	
LVIDS	Pearson Correlation Coefficient (r)	.524**	Positive correlation
	p-Value	<0.0001	Significant
	Number	80	

LVEF	Pearson Correlation Coefficient (r)	-.606**	Negative correlation
	p-Value	<0.0001	Significant
	Number	80	
SEVERITY OF CAD	Pearson Correlation Coefficient (r)	.297**	Positive correlation
	p-Value	.007	Significant
	Number	80	

In Total, the positive correlation was found in LVIDD vs NT pro-BNP and this correlation was statistically significant. In Total, the positive correlation was found in LVIDS vs NT pro-BNP and this correlation was statistically significant. In Total, the negative correlation was found in LVEF vs NT pro-BNP and this correlation was statistically significant. In Total, the positive correlation was found in severity of CAD vs NT pro-BNP and this correlation was statistically significant.

Case(n=50)		NT pro-BNP	Remarks
LVIDD	**Pearson Correlation Coefficient (r)**	.294*	Positive correlation
	p-Value	.038	Significant
	Number	50	
LVEF	**Pearson Correlation Coefficient (r)**	-.415**	Negative correlation
	p-Value	.003	Significant
	Number	50	
LVIDS	**Pearson Correlation Coefficient (r)**	.291*	Positive correlation
	p-Value	.041	Significant
	Number	50	
SEVERITY OF CAD	**Pearson Correlation Coefficient (r)**	.184	Positive correlation
	p-Value	.201	Not Significant
	Number	50	
Control(n=30)			
LVIDD	**Pearson Correlation Coefficient (r)**	.177	Positive correlation
	p-Value	.349	Not Significant
	Number	30	
LVEF	**Pearson Correlation Coefficient (r)**	-.270	Negative correlation
	p-Value	.149	Not Significant
	Number	30	
LVIDS	**Pearson Correlation Coefficient (r)**	.058	Positive correlation
	p-Value	.760	Not Significant
	Number	30	
SEVERITY OF CAD	**Pearson Correlation Coefficient (r)**	.049	Positive correlation
	p-Value	.796	Not Significant
	Number	30	

In case, the positive correlation was found in LVIDD vs NT pro-BNP and this correlation was statistically significant. In case, the positive correlation was found in LVIDS vs NT pro-BNP and this correlation was statistically significant. In case, the negative correlation was found in LVEF vs NT pro-BNP and this correlation was statistically significant. In case, the positive correlation was found in CAG vs NT pro-BNP and this correlation was not statistically significant.

In control, the positive correlation was found in LVIDD vs NT pro-BNP and this correlation was not statistically significant. In control, the positive correlation was found in LVIDS vs NT pro-BNP and this correlation was not statistically significant. In control, the negative correlation was found in LVEF vs NT pro-BNP and this correlation was not statistically significant. In control, the positive correlation was found in CAG vs NT pro-BNP and this correlation was not statistically significant.

Systolic heart failure group(n=40)		NT pro-BNP	Remarks
LVIDD	Pearson Correlation Coefficient (r)	.170	Positive correlation
	p-Value	.293	Not Significant
	Number	40	
LVEF	Pearson Correlation Coefficient (r)	-.303	Negative correlation
	p-Value	.058	Not Significant
	Number	40	
LVIDS	Pearson Correlation Coefficient (r)	.162	Positive correlation
	p-Value	.316	Not Significant
	Number	40	
SEVERITY OF CAD	Pearson Correlation Coefficient (r)	.081	Positive correlation
	p-Value	.618	Not Significant
	Number	40	
Diastolic heart failure group (n=10)			
LVIDD	Pearson Correlation Coefficient (r)	-.296	Negative correlation
	p-Value	.406	Not Significant

	Number	10	
LVEF	Pearson Correlation Coefficient (r)	-.102	Negative correlation
	p-Value	.779	Not Significant
	Number	10	
LVIDS	Pearson Correlation Coefficient (r)	-.329	Negative correlation
	p-Value	.354	Not Significant
	Number	10	
SEVERITY OF CAD	Pearson Correlation Coefficient (r)	.224	Positive correlation
	p-Value	.534	
	Number	10	

In systolic, the positive correlation was found in LVIDD vs NT pro-BNP and this correlation was not statistically significant. In systolic, the negative correlation was found in LVEF vs NT pro-BNP and this correlation was not statistically significant. In systolic, the positive correlation was found in LVIDS vs NT pro-BNP and this correlation was not statistically significant. In systolic, the positive correlation was found in CAG vs NT pro-BNP and this correlation was not statistically significant.

In Diastolic, the negative correlation was found in LVIDD vs NT pro-BNP and this correlation was not statistically significant. In Diastolic, the negative correlation was found in LVEF vs NT pro-BNP and this correlation was not statistically significant. In Diastolic, the negative correlation was found in LVIDS vs NT pro-BNP and this correlation was not statistically significant. In Diastolic, the positive correlation was found in CAG vs NT pro-BNP and this correlation was not statistically significant.

4. Discussion

Brain natriuretic peptide is a neurohormone synthesized in ventricular myocardium and released in response to cardiac stretch. NT-proBNP is the N-terminal fragment of the prohormone BNP. These natriuretic peptides have prognostic value across the full spectrum of acute coronary syndrome patients. Patients with elevated BNP or NT-proBNP are at significantly increased risk for subsequently developing heart failure. The NT proBNP seems to be affected more by worsening renal function than BNP. So patients with creatinine >2.0mg/Dl were excluded from the study.

In our study 80 patients of STEMI were involved of which 56 were males (70%) and 24 were females (30%). Mean age of presentation of STEMI was 57.02 yrs.

Sime manola et al[9]. in 2009 also found that mean age of STEMI presentation was 58.9± 10.3 and males were 74.5% and females were 25.5%.

Mean age of STEMI presentation of **Ragaa H.M. Salama et al[10].** study in 2011 was 60.72±0.9 and among the STEMI patients 77.77% were males and 22.23% were females.

In case, the mean CPK(mean± s.d.) of patients was 1570.9200± 1582.8160and in control, the mean CPK (mean± s.d.) of patients was 1473.2000± 1440.5551. The association of mean CPK vs two groups was not statistically significant (p=0.7831).

In case, the mean CPK-MB(mean± s.d.) of patients was 179.7600± 241.2701and in control, the mean CPK-MB (mean± s.d.) of patients was 126.8667± 116.6326. The association of mean CPK-MB vs two groups was not statistically significant (p=0.2651).

In our study the mean NT pro-BNP (mean± s.d.) of patients was 4206.2600± 3985.7472(pg/mL) in patients with LVEF <40%. In case, the mean NT pro-BNP (mean± s.d.) of patients was 4206.2600± 3985.7472(pg/Ml), and the mean NT pro-BNP (mean± s.d.) of patients was 382.8333± 176.8588(pg/mL) in patient with LVEF >40%.

Hanan Radwan et al[11]. in 2014 found that the mean NT pro-BNP(mean± s.d.) was 2569±2270.5 (pg/ml) in patient with LVEF <40% and the mean NT pro-BNP (mean± s.d.) was 328.4±46.8(pg/ml). In the present study we found that HTN was present in 52.5% of all STEMI patients while DM was present in 53.75% of all STEMI patients. Among all STEMI patients 57.5 % were smokers.

Wojciech Drewniak et al[12]. in 2015 showed that HTN was present in 65% of all STEMI patients.

Sime manola et al[10]. in 2009 found that among all STEMI patient 59.57% had HTN, 44.68% had DM, 53.19% of all STEMI patients were smokers.

Ragaa H.M. Salama et al[9]. in 2011 found that among all STEMI patients HTN and DM was present in 55.5% cases.
In the present study we found that STEMI patient presented with heart failure more common In case of AWSTEMI (82.0%) compared to IWSTEMI (18.0%).

Kang Q et al[13].(2017) found that levels of NT-proBNP in the extensive anterior wall infarction group were higher compared to that of the inferior wall infarction groups: $p < 0.05$; the levels of NT-proBNP in the inferior wall and posterior wall infarction group were higher compared with the inferior wall infarction group and anteroseptal wall infarction group: $p < 0.05$.
In all STEMI cases, the positive correlation was found in LVIDD vs NT pro-BNP and this correlation was statistically significant.

In all STEMI cases, the positive correlation was found in LVIDS vs NT pro-BNP and this correlation was statistically significant.

In all STEMI cases, the negative correlation was found in LVEF vs NT pro-BNP and this correlation was statistically significant.

Hanan Radwan et al[11]. in 2014 showed that the negative correlation was found in LVEF vs NT pro-BNP and this correlation was statistically significant.

In our study we found that in all patient with STEMI, the positive correlation was found in severity of CAD vs NT pro-BNP and this correlation was statistically significant.

We found that in patients with acute coronary syndrome, the number of vessels affected and percentage of stenosis were significantly higher statistically in those with high NT-proBNP (equal to or more than 300 pg/ml) compared to those with low NT-proBNP (less than 300 pg/ml). These results were concordant with other studies that focused on the association between the severityof CAD and NT-pro-BNP level.

In case, the positive correlation was found in LVIDD vs NT pro-BNP and this correlation was statistically significant. In case, the positive correlation was found in LVIDS vs NT pro-BNP and this correlation was statistically significant.

In case, the negative correlation was found in LVEF vs NT pro-BNP and this correlation was statistically significant. The underlying pathomechanism was not fully understood, but a direct release of NT proBNP from ischemic cardiomyocytes in addition to ischemia induced by increase in ventricular wall stress was postulated .

In case, the positive correlation was found in CAD vs NT pro-BNP and this correlation was not statistically significant.

In the present study, we found that the ejection fraction was significantly reduced in patients with NT-proBNP equal to or more than 700 pg/ml compared to patients with NT-proBNP less than 700 pg/ml. This result was comparable to that reported by Shahabi et al.. Furthermore, we found highly significant negative correlation between NT-proBNP and ejection fraction($r = 0.234$, $p = 0.0063$). This result was concordant with **Shahabi et al**[14]. and was also supported by **Emdin et al.**[15]who found that NT-proBNPhad acceptable accuracy for identifying heart failure due to left ventricular dysfunction.

Kang Q et al.[13] **in 2017** found that the levels of NT-proBNP in the multi-vessel group were higher than those in the single-vessel group: $p < 0.05$. The BNP level was positively correlated with age, heart rate, creatinine kinase-myocardial band (CK-MB), cardiac troponin T (cTnT), whereas it was negatively correlated with left ventricular ejection fraction (LVEF).

The NT proBNP is a powerful biomarker for the diagnosis and prognosis of HF. It is elevated in conditions of increased ventricular wall stress and is most commonly used to rule out HF in dyspnoeic patients.

Rao SJ et al [16] **(2016)** found that early identification of heart failure as a post-myocardial infarction is very important in a clinical setting. A study was planned to determine the levels of (N-terminal) NT-pro-(Brain Natriuretic Peptide) BNP in systolic heart failure consequential to ischaemic heart disease, and to find out the risk factors in those patients. The levels of NT-pro-BNP was determined in 100 patients admitted to Basaveshwar Teaching and General Hospital, Kalaburagi, with a diagnosis of systolic heart failure having an ejection fraction<40% subsequent to ischaemic heart disease. The levels of NT-pro-BNP ranged between 358 pg/ml and 3000 pg/ml with a mean value of 2049 pg/ml and a median value of 1886 pg/ml. NT-pro-BNP level had a good predictive value for heart failure (p-0.03).

Arafath MY et al [17] **(2019)** found that Cardiac enzymes (Troponin T and CKMB) was elevated for the majority of the patients (N=27, 67.5%). Cardiac enzymes (Troponin T and CKMB) were normal for only 32.5% of the patients. Even though the study is done in patients without clinical signs of heart failure, the levels of NT-proBNP had an inverse relationship with Ejection Fraction. Low NT-proBNP levels at the time of admission rule out high-risk patients or patients with heart failure.

Ozturk TC et al [18] **(2011)** found that NT-proBNP levels were significantly higher in hospitalized patients compared to outpatients, and this finding was correlated with the clinical status of the patients. The mean NT-proBNP value of the patients was 9741.9 ± 8973 pg/ml (range: 245-35000) while the mean NT-proBNP value of patients diagnosed with non-decompensated congestive heart failure was 688.9 ± 284.5 pg/ml (range: 115-1450.65). NT-proBNP can be used as an easy diagnostic method for congestive heart failure.

Kashlov JK et al [19] **(2016)** found that all patients with STEMI and elevated serum levels of NT- proBNP have left ventricular ejection fraction <50%. Their results imply that NT –proBNP level and its increase in the serum may be used as a biomarker for the severity of the ischemic heart disease.

Our study was designed to assess the diagnostic value of plasma NT proBNP level as a non-invasive indicator of LV dysfunction and to differentiate it from other causes of dyspnoea in background of STEMI. Also, to correlate the NT proBNP values with echocardiographic ejection fraction.

Although echocardiography is considered the gold standard for the detection of LV dysfunction, it is expensive, not easily accessible, and may not always reflect an acute condition. In our study NT proBNP levels correlated well with reduced LVEF. Patients with a final diagnosis of LV dysfunction had significantly higher levels of NT proBNP than those without LV dysfunction (P <0.001).

5. Summary

1) In the present study 56 males (70%) and 24 females (30%) were involved and male and female ratio was 2.33:1.
2) Present study showed that males were more commonly affected by STEMI compared to female and STEMI was also more predominant in smokers.
3) We found thatthe heart failure group of subjects had mean CPK and CPK-MB higher in comparison to that in the group without heart failure.

4) It was found that NT pro-BNP was significantly increased in heart failure group compared to group without heart failure.
5) Our study found that LVIDD and LVIDS were higher in heart failure group compared to group without heart failure which was statistically significant. Mean ($^{+}$/- SD)ejection fraction was significantly lower in heart failure group.
6) Heart failure was more common in the patients with AWSTEMI compared to IWSTEMI group and it was statistically significant.
7) Heart failure was more common in the patients with triple vessel coronary artery disease and it was statistically significant.
8) All patients of AMI with and without heart failure taken together(n=80) when studied it was found that the NT pro-BNP was positively correlated with LVIDD, LVIDS and severity of CAD in STEMI patients which was statistically significant. NT pro-BNP was negatively correlated with LVEF in STEMI patient and it was statistically significant.
9) In patients of AMI with systolic heart failure group only (n=40) NT pro-BNP was positively correlated with LVIDD, LVIDS and severity of CAD but NT pro-BNP was negatively correlated with LVEF which were not statistically significant.
10) In patients of AMI with diastolic heart failure only (n=10) NT pro-BNP was positively correlated with LVIDD, LVIDS and severity of CAD .It was also observed that NT pro-BNP was negatively correlated with LVEF though that was not statistically significant.
11) It seems that the NT-proBNP in acute coronary syndrome may be a very useful marker. There is a positive correlation between NT Pro BNP and the number of coronary artery (ies) involved and the severity of luminal stenosis. Last but not the least NT Pro BNP is a very valuable marker for predicting higher incidence of heart failure and lower ejection fraction.

Abbreviations

NT pro-BNP-N-Terminal pro B type natriuretic peptide
STEMI-ST elevation myocardial infarction
ACS= acute coronary syndrome
PCI= percutaneous coronary intervention
CABG= coronary artery bypass graft
CHF= congestive heart failure
HTN-Hypertension
DM-Diabetes mellitus
COPD-Chronic obstructive pulmonary disease
PND-Paroxysmal nocturnal dyspnoea
BP-Blood pressure
HR-Heart rate
RR-Respiratory rate
JVP-Jugular venous pulse
HB-Hemoglobin
TLC-Total leucocyte count
CPK -Creatine phosphokinase
CPK-MB- Creatine phosphokinase-Muscle band
LVIDD-Left ventricular internal diameter in diastole
LVIDS- Left ventricular internal diameter in systole
LVEF-Left ventricular ejection fraction
RWMA-Regional wall motion abnormality
LMCA-Left main coronary artery
CAG – Coronary Angiography
LAD-Left anterior descending artery
LCX-Left circumflex artery
RCA-Right coronary artery
M ± SD= mean ± standard deviation
N-No
Y-Yes
R-Raise
NR-Not raise

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