Registry Study of Clinical Data and in Hospital Outcome in Patients Admitted with Acute Coronary Syndrome at Tanta University Hospital in 2019

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Authors' contributions

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ABSTRACT

Background: A quick but thorough assessment of the patient's history and findings on physical examination, electrocardiography, and cardiac biomarker tests permit accurate diagnosis and aid in early risk stratification. This work aimed to analyze the diagnostic and prognostic tools, the modalities of management, and the hospital outcome of patients with acute coronary syndrome (ACS) at Tanta University Hospital in one year.
Methods: This ACS registry at Tanta university hospital is a prospective observational registry for 200 consecutive admitted patients with proven ACS from January 2019 to January 2020.
Results: A higher percent of hypertension, family history of ischemic heart disease and SCD, previous history of chronic kidney disease (CKD), and lower percent of a previous history of IHD in STEMI compared to NSTEMI/UA. In-hospital death, in-hospital reinfarction, and reduced ejection fraction are higher in STEMI than in NSTEMI/UA patients. (P value = 0.015, 0.018 and 0.001 respectively) without significant differences regarding in-hospital congestive heart failure (CHF) and ischemic stroke. History of CKD, higher Killip class, and in-hospital stroke were independently

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affecting in-hospital mortality. Also, the history of higher Killip class was independently affecting in-hospital reinfarction and in-hospital CHF. Old age and occurrence of in-hospital reinfarction were independently affecting in-hospital stroke.

Conclusion: Hypertension, diabetes, dyslipidemia, and smoking are the major risk factors for ACS so, controlling these risk factors will improve in-hospital outcomes. In STEMI, most patients underwent PPCI, which was reflected in the outcome. In NSTEMI/UA patients, both conservative and invasive management was done, taking into consideration the risk stratification of each patient, making management easier and with a good outcome.

1. INTRODUCTION

The term acute coronary syndrome (ACS) refers to any group of clinical symptoms compatible with acute myocardial ischemia and includes unstable angina (UA), non–ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). These high-risk manifestations of coronary atherosclerosis are important causes of the use of emergency medical care [1].

A quick but thorough assessment of the patient's history and findings on physical examination, electrocardiography, and cardiac biomarker tests permit accurate diagnosis and aid in early risk stratification, which is essential for guiding treatment [2].

Coronary artery disease (CAD) is a leading cause of morbidity and mortality in developed and developing countries [3].

The incidence and outcomes of ACS depend upon population exposure to risk factors, access to quality health care services, and health-seeking behavior of the community [4-6]. These, in turn, are influenced by socioeconomic status, access to health information, geographical characteristics, and cultural practices. Thus, the characteristics, the treatment, and outcomes are likely to vary in different parts of the country and between the different countries [4,7].

High-risk patients with UA/NSTEMI are often treated with an early invasive strategy involving cardiac catheterization and prompt revascularization of viable myocardium at risk [7].

Tanta university hospital is a percutaneous coronary intervention (PCI) capable center, so cardiac catheterization can be done in the form of primary PCI, pharmacoinvasive PCI, and rescue PCI.

Clinical outcomes can be optimized by revascularization, coupled with aggressive medical therapy that includes anti-ischemic, antiplatelet, anticoagulant, and lipid-lowering drugs [4].

Evidence-based guidelines provide recommendations for the management of ACS. However, therapeutic approaches to the management of ACS continue to evolve at a rapid pace driven by a multitude of large-scale randomized controlled trials [7].

This work aimed to analyze the diagnostic and prognostic tools, the modalities of management, and the in-hospital outcome of patients with ACS at Tanta University Hospital in one year.

2. SUBJECTS AND METHODS

This ACS registry at Tanta university hospital is a prospective observational registry for all consecutive patients with proven ACS that are admitted at Tanta university hospital. The study had been run for 12 months from January 2019 to January 2020, during which data collection and follow up have been done for 200 case.

Our study involving the following phases:

Phase I: Baseline measurement of process of care and health care services, standard case record form (registry form) has been filled out for each patient during admission time.

Phase II: Follow up assessment for morbidity and mortality during the hospital stay.

Data were collected according to standard case record form, including clinical and demographic characters of included patients, course during the stay, diagnostic and therapeutic options offered for those patients during the hospital stay and upon discharge.

Our registry included the following data:

1) Full history taking with an emphasis on age, sex, history of risk factors for CAD as: Diabetes Mellitus, hypertension, smoking, and past history and family history of CAD, history of chronic kidney disease (CKD), and previous history of ischemic heart disease (IHD).

2) Full clinical examination: vital signs (heart rate, blood pressure, and respiratory rate), general examination (with attention to height, weight, body mass index, patient look, decubitus, cyanosis, jaundice, with special attention to signs of heart failure) and local cardiac examination (abnormal pulsation, heart sounds & murmurs).

3) Standard 12-lead ECG was obtained within 10 minutes of first medical contact (FMC) according to ESC guidelines 2017 including: (limb leads I, II, III, aVR, aVL, aVF, and chest leads from V1 to V6) for all patients on admission to the hospital [8]. Right pericardial leads (V3R, V4R, V5R, V6R) and posterior chest leads (V7 to V9) were done for some patients to detect the posterior wall and right ventricular infarction [8].

4) Routine laboratory investigation including CKMB/Troponin (to differentiate between types of ACS; STEMI NSTEMI or UA), fasting and 2 hours postprandial blood sugar level, total cholesterol, LDL, HDL, triglycerides, urea & creatinine level, liver function tests, and complete blood count

5) Echocardiography: All studies were performed using (a GE vivid seven Cardiac ultrasound phased array system with tissue Doppler imaging using M4S transducer 4 M.HZ.). Two- Dimensional echocardiographic assessment by M-mode was done during admission after successful PCI.

6) Modality of treatment offered to the patient either conservative medical treatment in patients with late symptoms more than 48 hours of chest pain, thrombolytic therapy or invasive modalities e.g. (1ry PCI, pharmacoinvasive PCI or rescue PCI) as regard STEMI patient.
As regards NSTEMI or UA patient management was in the form of conservative medical management, or it may need reperfusion in special situations according to validated risk scores.
Reperfusion either through: a) Primary percutaneous intervention for Infarct related artery (IRA) b) Pharmacoinvasive technique: patients receive thrombolytic

therapy followed by coronary angiography either immediately in case of failed thrombolytic or within 3-24 hours after a sign of successful reperfusion [8]. The used type of thrombolytic in Tanta university hospital CCU is streptokinase due to low cost and availability in CCU.

7) In-hospital outcome detection: a) In-hospital reinfarction, b) In-hospital congestive heart failure (CHF) c) Ischemic stroke: Detection of stroke occurrence especially in the first few hours after symptoms in both STEMI and Non-STEMI. This was discovered by occurrence of neurological manifestations and supported by imaging as CT brain or MRI. d) In-hospital death: Detection of the patient who died during hospital admission in correlation with different risk factors.

2.1 Statistical Analysis

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) Qualitative data were described using number and percent. The Kolmogorov-Smirnov test was used to verify the normality of distribution Quantitative data were described using range (minimum and maximum), mean, standard deviation, median and interquartile range (IQR). Univariate and multivariate logistic regression analysis were done. The significance of the obtained results was judged at the 5% level.

3. RESULTS

Males were more common than females representing 67% of ACS patients (134 patients) while females representing 33% (66 patients). In (NSTEMI/ UA): 76% were males (79 patients) compared to STEMI: males represented 57.3% (P = 0.005). STEMI patients has younger age at presentation than (NSTEMI/UA) patients (P value<0.001) (Table 1).

There was statistically significant difference between both STEMI and (NSTEMI/UA) patients with higher incidence of hypertension in STEMI group with P value 0.012. There was statistically significant difference between STEMI and (NSTEMI/UA) regarding family history as STEMI has higher incidence of family history of IHD and SCD P value <0.001). There was statistically significant difference between STEMI and NSTEMI/UA patients regarding previous history of IHD as a risk factor as it was more frequent in

NSTEMI/UA than STEMI patients. (P value <0.001). There was statistically significant difference between STEMI and NSTEMI/UA patients regarding previous history of CKD as a risk factor as it was more frequent in STEMI than NSTEMI/UA patients. (P value <0.041). There was no statistically significant difference between STEMI and (NSTEMI/UA) patients regarding diabetes, smoking, dyslipidemia as risk factor (P value=0.476) (Table 1).

There was no statistically significant difference between STEMI and NSTEMI/UA patients regarding systolic blood pressure, diastolic blood pressure at presentation. Pulse was higher in NSTEMI/UA than in STEMI patients. (P value =0.005). Killip class as STEMI patients presented with higher Killip class ≥2. (P value =0.019) (Table 1).

There was no statistically significant difference between STEMI and NSTEMI/UA patients regarding fasting blood glucose level, 2 hours postprandial blood glucose level, HDL, LDL. cholesterol, triglycerides levels, WBCs count, platelet count, creatinine, SGPT and SGOT. Hb level with higher levels in NSTEMI/UA (P value <0.001). CKMB level with higher levels of CKMB in STEMI than NSTEMI/UA patients. (P value <0.001) (Table 2).

In this study anterior STEMI represented 44.8% of all STEMI patients (43 patients). Inferior STEMI represented 41.7% of patients (40 patients) while lateral STEMI represented 13.5% of patients (13 patients).

It was found that in STEMI 71.9% of patients underwent PCI (69 patients) while thrombolytic therapy was given in 12.5% of patients (12 patients) and conservative medical treatment was given in 15.6% of patients (15 patients). In NSTEMI conservative medical therapy was the most common strategy 66.7% of (38 patients) while 33.3% of patients underwent PCI (19 patients). In UA: conservative medical therapy was the most common strategy 78.7% of (37 patients) while 21.3% of patients underwent PCI (10 patients). There was statistically significant difference between STEMI, NSTEMI and UA in modality of treatment as PCI was frequently done in STEMI more than NSTEMI and UA in contrast conservative medical treatment was frequently used in UA more than STEMI and NSTEMI.(P value <0.001).
Distribution of the studied cases according to different parameters regarding PCI is shown in Table 3.

In-hospital death, in-hospital reinfarction EF in STEMI patients is lower than in NSTEMI/UA patients. (P value = 0.015, 0.018 and 0.001 respectively). There was no statistically significant difference between STEMI and NSTEMI/UA regarding in-hospital CHF and ischemic stroke. (Table 5)

It was found in univariate that history of CKD, higher Killip class, occurrence of ischemic stroke and higher creatinine ratio during admission were associated with higher risk of in-hospital death. Also, STEMI was associated with higher risk of in-hospital death. In multivariate analysis history of CKD higher Killip class and in-hospital stroke were independently affecting in-hospital mortality. In-hospital death increased three times in patients with history of CKD, eight times in patients developed ischemic stroke during admission and 13 times for patients with higher Killip class. Higher HDL level was found to be protective against in-hospital death. (Table 6)

It was found in univariate analysis that history of higher Killip class, anterior and lateral STEMI were associated with higher risk of in-hospital reinfarction. In multivariate analysis history of higher Killip class and lateral STEMI were independently affecting in-hospital re infarction. In-hospital reinfarction occurred nine times in patients with higher Killip class eight times in patients with lateral STEMI. Higher platelet count was found to be protective against in-hospital reinfarction. (Table 7)

It was found in univariate analysis that history of dyslipidemia, CKD, higher Killip class, higher pulse and higher level of 2 hours post prandial glucose level, LDL, cholesterol, CKMB and creatinine during admission were associated with higher risk of in-hospital CHF. In multivariate analysis history of higher Killip class and lateral STEMI were independently affecting in-hospital CHF. CHF incidence was fifteen times in patients with higher Killip class eighteen times in patients with high creatinine ratio and twice for higher level CKMB and LDL. Higher HDL level was found to be protective against in-hospital CHF. (Table 8)

It was found in univariate analysis that old age, lower ejection fraction and in-hospital reinfarction were associated with higher risk of in-hospital stroke. In multivariate analysis old age and occurrence of in-hospital reinfarction were independently affecting in-hospital stroke (Table 9).

4. DISCUSSION

Clinical records are an excellent opportunity to evaluate the clinical presentation, behavior, treatment and outcome of a disease and the patients affected by it. Randomized clinical trials, while also providing clinical information, follow specific inclusion criteria, thus limiting the sample. In registries, patients are not selected, and their findings more properly reflect the so-called "real world", in which cardiologists work and live their daily routine.

In this study 101 patients were diabetic 50.5% in all study population this is similar to study done by Tillin et al. [9] more than 40% of patients with ACS have DM. Additionally in study done by Arnold et al [10] showed that mortality in patients with ACS is 2-3 fold elevated in diabetic patients compared with non diabetic ones.

In this study there was no statistically significant difference between STEMI and (NSTEMI/UA) patients regarding diabetes as risk factor.

Diabetes is an important risk factor which was found in study done by Mohanan et al. [11],

reporting 35.5% having DM/ Interestingly, DM did not show a significant association with the type of ACS.

This is similar to data stated in RENASICA III registry [12], that showed no statistically significant difference between STEMI and NSTEMI/UA as regard diabetes as risk factor.

In this study 92 were hypertensive representing 46% of all study population. There was statistically significant difference between both STEMI and (NSTEMI/UA) patients with higher incidence of hypertension in STEMI group this may be due to increase incidence of hypertension in Egyptian population even in young age.

Similarly, a study done by Mohanan et al. [11] stated that hypertension has significantly higher prevalence in STEMI than NSTEMI/UA patients.

In contrast to our study, a study done by Carlo et al. [1] stated that hypertension was statistically significant higher in NSTEMI/UA than STEMI patients.

Table 1. Demographic data, risk factors, clinical examination and Killip class of ACS patients

		Total (n = 200)		STEMI (n = 96)		NSTEMI/UA (n = 104)		Test	p
Age (years)		54.41 ± 11.74		11.87 ± 51.01		57.54 ± 10.76		t= 4.079[*]	<0.001[*]
		N	%	N	%	N	%		
Sex	Male	134	67.0	55	57.3	79	76.0	χ^2=7.87[*]	0.005[*]
	Female	66	33.0	41	42.7	25	24.0		
Risk factors of ACS									
Hypertension		92	46.0	53	55.2	39	37.5	χ^2=6.302	0.012[*]
Diabetes		101	50.5	51	53.1	50	48.1	χ^2=0.509	0.476
Smoking		104	52.0	44	45.8	60	57.7	χ^2=2.813	0.094
Dyslipidemia		122	61.0	55	57.3	67	64.4	χ^2=1.067	0.302
Family history		71	35.5	46	47.9	25	24.0	χ^2=12.431[*]	<0.001[*]
History of IHD		75	37.5	17	17.7	58	55.8	χ^2=30.855[*]	<0.001[*]
History of CKD		29	14.5	19	19.8	10	9.6	χ^2=4.170[*]	0.041[*]
Clinical examination									
Systolic blood pressure (mmHg)		131.8 ± 29.03		30.12 ± 134.9		129.0 ± 27.83		T = 1.432	0.154
Diastolic blood pressure (mmHg)		81.90 ± 15.58		15.55 ± 83.44		80.48 ± 15.54		T = 1.344	0.181
Pulse (b/pm)		84.69 ± 16.04		10.21 ± 81.49		87.64 ± 19.56		T = 2.820[*]	0.005[*]
Killip class		1.0 (1.0 – 1.0)		(2.0 – 1.0) 1.0		1.0 (1.0 – 1.0)		U= 4296[*]	0.019[*]

*Data are presented as mean ± SD, number (and %) or median (IQR). ACS: Acute coronary syndrome, IHD: Ischemic heart diseases, CKD: Chronic kidney disease, χ^2: Chi square test, t: Student t-test, U: Mann Whitney test, p: p value for comparing between the studied groups, *: Statistically significant at p ≤ 0.05*

Table 2. Laboratory data in ACS patients

	Total (n = 200)	STEMI (n = 96)	NSTEMI/UA (n = 104)	Test	p
Blood glucose					
Fasting blood glucose(mg/dl)	127.5(85–150)	132.5(85–150)	120.00(80–150)	U= 4592.5	0.327
2hrs post prandial blood sugar(mg/dl)	140(120–250)	200.0(120–290)	140 (120–230)	U= 4262.5	0.074
Lipid profile					
HDL (mg/dl)	36.63 ± 10.44	36.35 ± 11.15	36.88 ± 9.79	**t** =0.352	0.725
LDL (mg/dl)	144.62 ± 29.24	144.79 ± 29.11	144.46 ± 29.49	t=0.080	0.937
Cholesterol (mg/dl)	216.88 ± 55.49	218.52 ± 56.94	215.37 ± 54.35	t=0.401	0.689
Triglycerides (mg/dl)	162.45 ± 47.04	159.11 ± 45.66	165.53 ± 48.29	0.963	0.337
Complete blood count					
Hemoglobin (mg/dl)	12.88 ± 1.51	12.43 ± 1.31	13.30 ± 1.56	t=4.244[*]	<0.001[*]
White blood cells (cell/cubic mm×10^3)	12.19 ± 7.74	14.27 ± 9.22	10.28 ± 5.45	U=4238.5	0.065
Platelets (cell/cubic mm×10^3)	239.75 ± 71.10	237.82 ± 71.65	241.53 ± 70.90	U=4775	0.596
Cardiac enzymes					
CK-MB (IU/L)	57.0(34.5 – 151.5)	124.5(48 – 195.5)	42.0(27.0 – 121.5)	U= 3053[*]	<0.001[*]
Kidney and Liver functions					
Creatinine (cr) (mg/dl)	1.10(1.0 – 1.4)	1.10(1.0 – 1.4)	1.10(1.0 – 1.4)	U=4871.00	0.757
SGPT (U/L)	43.0(23.0 – 64.5)	43.0(23.0 – 63.5)	43.0(25.5 – 65.0)	U=4638.5	0.387
SGOT (U/L)	56.0(28.0 – 150.0)	54.0(30.0 – 147.5)	85.0(28.0 – 151.0)	U=4856	0.739

While 104 were active smokers this came in agreement with a study conducted by Chow et al [13] smoking has a strong pro-thrombotic effect, and smoking cessation is potentially the most (cost) effective of all secondary prevention measures [13].

It is noteworthy that Baker et al [14] stated that an effective smoking treatment requires "chronic care", a term that strongly underlines the huge difficulty of effectively and permanently quitting smoking [14].

However in our study there was no statistically significant difference in smoking between STEMI and NSTEMI/UA this similar to data obtained from study done by Deora et al [15] stated that no difference between STEMI and NSTEMI/UA patients regarding smoking.

61% of all study population were found to have history of dyslipidemia which is large portion of population but there was no statistically significant difference between both STEMI and NSTEMI in incidence of dyslipidemia. Higher incidence of dyslipidemia may be due to

hereditary factors and mostly due to bad dietary habits.

This is similar to study done by Montalescot et al [16] that also showed very high incidence of dyslipidemia among all ACS patients but no statistically significant difference between STEMI and NSTEMI/UA patients.

Family history was found to be more in STEMI patients in our study it may be due to younger age in this group than NSTEMI/UA group family history includes family history of IHD or sudden cardiac death.

Similarly, study done by Sultana et al [17] showed increase incidence of family history in STEMI patients more than NSTEMI/UA patients.

In contrast to study done by Ralapanawa et al [18] stated that no statistically significant difference between STEMI and NSTEMI/UA regarding presence of family history of ACS or SCD.

Table 3. Distribution of the studied cases according to different variables in STEMI group regarding PCI (n = 75), NSTEMI group (n = 19) and UA group (n = 10)

	STEMI		NSTEMI group (n = 19)		UA group (n = 10)	
	No.	%	No.	%	No.	%
Infarction related artery						
LAD	34	45.3	12	63.2	8	80.0
LCX	14	18.7	2	10.5	1	10.0
RCA	27	36.0	5	26.3	1	10.0
Door to balloon (min) (n = 68)						
Median (IQR)	33.0(20.0 – 40.0)		----		----	
No. of diseased vessels						
Single vessel	44	58.7	9	47.4	5	50.0
Multi vessel	31	41.3	10	52.6	5	50.0
Type of intervention						
DES	67	89.3	16	84.2	8	80.0
BMS	3	4.0	1	5.3	1	10.0
Balloon angioplasty	1	1.3	0	0.0	1	10.0
Differed stenting	4	5.3	2	10.5	0	0.0
Final TIMI flow						
<3	12	16.0	3	15.8	0	0.0
3	63	84.0	16	84.2	10	100.0

LAD: left anterior descending LCX: left circumflex artery RCA: right coronary artery
DES: drug eluting stent. BMS: bare metal stent

Table 4. Distribution of the studied cases according to thrombolytic therapy (n = 12)

Thrombolytic therapy	No.	%
Failed	**5**	**41.7**
Rescue PCI	3	25.0
Died	2	16.7
Successful	**7**	**58.3**
Pharmacoinvasive	3	25.0
Medical treatment	4	33.3

Table 5. In-hospital outcome and ejection fraction in ACS patients

In hospital	Total (n = 200)		STEMI (n = 96)		NSTEMI/UA (n = 104)		χ^2	p
	No.	%	No.	%	No.	%		
In-hospital outcome								
Death	29	14.5	20	20.8	9	8.7	5.973[*]	0.015[*]
Reinfection	14	7.0	11	11.5	3	2.9	5.637[*]	0.018[*]
CHF	31	15.5	14	14.6	17	16.3	0.118	0.731
Stroke	12	6.0	6	6.3	6	5.8	0.020	0.886
Echo (EF%)								
Mean ± SD	48.12 ± 10.43		45.50 ± 10.86		50.55 ± 9.44		T=3.516[*]	0.001[*]

Data are presented as mean ± SD or number (and %). CHF: congestive heart failure, EF: Ejection fraction,
*χ^2: Chi square test, t: Student t-test, p: p value for comparing between the studied groups, *: Statistically significant at p ≤ 0.05*

Table 6. Univariate and multivariate logistic regression analysis for the variables affecting in hospital death (n = 29/200) for total sample

	Univariate		#Multivariate	
	p	OR (95% C.I)	P	OR (95% C.I)
Age	0.193	1.023 (0.989 – 1.058)		
Hypertension	0.064	2.145 (0.955 – 4.816)		
History of CKD	0.009*	3.397 (1.361 – 8.479)	0.131	3.084 (0.715 – 13.300)
Reinfection	0.133	2.576 (0.750 – 8.846)		
CHF	0.170	1.949 (0.751 – 5.058)		
Stroke	0.011*	4.881 (1.434 – 16.615)	0.009*	8.540 (1.698 – 42.937)
Killip class (≥2)	<0.001*	11.096 (4.624 – 26.628)	<0.001*	13.648 (4.658 – 39.987)
Echo	0.778	0.995 (0.958 – 1.033)		
Systolic blood pressure (mmHg)	0.161	0.989 (0.975 – 1.004)		
Diastolic blood pressure (mmHg)	0.109	0.978 (0.951 – 1.005)		
Pulse (b/pm)	0.851	1.002 (0.978 – 1.027)		
Fasting blood glucose (mg/dl)	0.506	1.003 (0.994 – 1.012)		
2hrs post prandial blood sugar (mg/dl)	0.066	1.004 (1.000 – 1.008)		
Hemoglobin (mg/dl)	0.334	0.875 (0.667 – 1.147)		
WBCs (cell/cubic mm×10³)	0.059	1.046 (0.998 – 1.096)		
Platelets (cell/cubic mm×10³)	0.808	0.999 (0.994 – 1.005)		
HDL (mg/dl)	0.002*	0.931 (0.890 – 0.974)	0.001*	0.897 (0.843 – 0.956)
LDL (mg/dl)	0.577	0.996 (0.983 – 1.010)		
Cholesterol (mg/dl)	0.958	1.000 (0.993 – 1.007)		
Triglycerides (mg/dl)	0.710	0.998 (0.990 – 1.007)		
Creatinine (mg/dl)	0.018*	3.496 (1.239 – 9.865)	0.486	1.836 (0.333 – 10.130)
CK-MB (IU/L)	0.862	1.000 (0.998 – 1.002)		
SGPT (U/L)	0.507	1.002 (0.996 – 1.008)		
SGOT (U/L)	0.829	0.999 (0.995 – 1.004)		
PCI management	0.132	1.861 (0.830 – 4.176)		
Thrombolytic therapy management	0.069	3.260 (0.914 – 11.631)		
STEMI	0.017*	2.778 (1.196 – 6.451)	0.779	0.836 (0.238 – 2.930)

*OR: Odd`s ratio, C.I: Confidence interval, LL: Lower limit, UL: Upper Limit, #: All variables with p<0.05 was included in the multivariate, *: Statistically significant at p ≤ 0.05*

History of IHD was found in 37.7% of all ACS population and it was found that it's significantly higher in NSTEMI/UA than STEMI patients.

This is similar to study done by Carlo et al [19] showed that history of IHD was more frequent in NSTEMI/UA than STEMI patients.

History of CKD 14.5% of study population had history of CKD but there was increase in incidence in STEMI patients than in NSTEMI/UA patients.

In this study patients with STEMI were found to have higher killip class than (NSTEMI /UA)patients in contrast to RENASICA III registry [19] that stated that number of patients with higher killip class (more than 2) was significantly higher in NSTEMI/UA patients than STEMI patients.

Similarly, in a study done by Xavier et al [20] it was noted that STEMI patients presented with higher Killip class than (NSTEMI/UA) patients [20].

In our registry the group with the least ejection fraction is the STEMI group with mean 45% and there was statistically significant decrease in EF of STEMI patients than NSTEMI/UA patients.

Similarly a study done by Deora et al [15] stated that the left ventricular ejection fraction was significantly lower in the STEMI group than that in the NSTEMI/UA group.

Baker et al (2016) stated that an effective smoking treatment requires "chronic care", a term that strongly underlines the huge difficulty of effectively and permanently quitting smoking. This may be due to more extensive wall motion abnormalities in STEMI than NSTEMI also necrosis occurs more in STEMI in comparison to NSTEMI/UA.

In our study in-hospital death was significantly higher in STEMI than NSTEMI/UA patients.

This is similar to data in a study done by Azman et al [21] stated that higher mortality rates occur in STEMI than NSTEMI/UA patients. This may be due to multiple complications that may occur in STEMI patients and complication of treatment either thrombolytic therapy or PCI.

Table 7. Univariate and multivariate logistic regression analysis for the variables affecting in hospital re Infarction (n = 14/200) for total sample

	Univariate		#Multivariate	
	P	OR (95%C.I)	p	OR (95%C.I)
Age	0.120	1.038 (0.990 – 1.087)		
Smoking	0.214	0.488 (0.158 – 1.512)		
In-Hospital death	0.133	2.576 (0.750 – 8.846)		
Killip class (/≥2)	<0.001[*]	7.766 (2.451 – 24.609)	0.009[*]	9.607 (1.772 – 52.073)
Echo	0.724	1.009 (0.958 – 1.064)		
Systolic blood pressure (mmHg)	0.956	0.999 (0.981 – 1.018)		
Diastolic blood pressure (mmHg)	0.343	1.017 (0.983 – 1.052)		
Pulse (b/pm)	0.871	1.003 (0.970 – 1.037)		
Fasting blood glucose (mg/dl)	0.283	0.993 (0.979 – 1.006)		
2hrs post prandial blood sugar(mg/dl)	0.738	0.999 (0.993 – 1.005)		
Hemoglobin (mg/dl)	0.806	0.955 (0.663 – 1.377)		
WBCs (cell/cubic mm×10^3)	0.157	1.046 (0.983 – 1.113)		
Platelets (cell/cubic mm×10^3)	0.042[*]	0.990 (0.980 – 1.000)	0.016[*]	0.973 (0.952 – 0.995)
HDL (mg/dl)	0.298	1.027 (0.977 – 1.080)		
LDL (mg/dl)	0.817	1.002 (0.984 –.984)		
Cholesterol (mg/dl)	0.272	0.994 (0.984 – 1.004)		
Triglycerides (mg/dl)	0.425	0.995 (0.983 – 1.007)		
Creatinine (mg/dl)	0.522	0.522 (0.071 – 3.822)		
CK-MB (IU/L)	0.343	0.997 (0.990 – 1.003)		
SGPT (U/L)	0.148	1.005 (0.998 – 1.012)		
SGOT (U/L)	0.408	0.997 (0.990 – 1.004)		
STEMI location				
Anterior	0.033[*]	0.102 (0.013 – 0.835)	0.063	0.073 (0.005 – 1.153)
Lateral	0.003[*]	8.021 (1.993 – 32.278)	0.029[*]	8.797 (1.247 – 62.069)
Door to balloon (min)	0.547	1.012 (0.973 – 1.052)		

*OR: Odd`s ratio, C.I: Confidence interval, LL: Lower limit, UL: Upper Limit, #: All variables with p<0.05 was included in the multivariate, *: Statistically significant at p ≤ 0.05*

Table 8. Univariate and multivariate logistic regression analysis for the variables affecting in hospital CHF (n = 31/200) for total sample

	Univariate		#Multivariate	
	P	**OR (95% C.I)**	**P**	**OR (95% C.I)**
Age	0.659	1.007 (0.975 – 1.041)		
Hypertension	0.285	1.522 (0.705 – 3.286)		
Smoking	0.133	1.840 (0.831 – 4.075)		
Dyslipidemia	0.007*	3.954 (1.448 – 10.795)	0.718	1.406 (0.222 – 8.922)
History of IHD	0.176	1.703 (0.787 – 3.684)		
History of CKD	0.004*	3.759 (1.541 – 9.169)	0.416	1.965 (0.386 – 9.997)
Killip class (/≥2)	<0.001*	9.120 (3.944 – 21.088)	<0.001*	15.844 (4.434 – 56.608)
Echo	0.706	1.007 (0.971 – 1.045)		
Systolic blood pressure (mmHg)	0.209	0.991 (0.977 – 1.005)		
Diastolic blood pressure (mmHg)	0.539	0.992 (0.967 – 1.017)		
Pulse (b/pm)	0.031*	1.026 (1.002 – 1.050)	0.072	1.035 (0.997 – 1.075)
Fasting blood glucose (mg/dl)	0.078	1.008 (0.999 – 1.017)		
2hrs post prandial blood sugar(mg/dl)	0.045*	1.004 (1.000 – 1.008)	0.919	1.000 (0.994 – 1.007)
Hemoglobin (mg/dl)	0.437	1.105 (0.859 – 1.420)		
WBCs (cell/cubic mm×10³)	0.335	1.023 (0.976 – 1.072)		
HDL (mg/dl)	0.022*	0.954 (0.916 – 0.993)	0.042*	0.933 (0.873 – 0.998)
LDL (mg/dl)	0.001*	1.025 (1.011 – 1.040)	0.041*	1.033 (1.001 – 1.065)
Cholesterol (mg/dl)	0.005*	0.989 (0.982 – 0.997)	0.001*	0.980 (0.968 – 0.992)
Triglycerides (mg/dl)	0.425	1.003 (0.995 – 1.012)		
Creatinine (mg/dl)	<0.001*	7.311 (2.531 – 21.117)	0.006*	18.516(2.322 – 147.621)
CK-MB (IU/L)	0.004*	1.003 (1.003 – 1.004)	0.001*	1.004 (1.002 – 1.007)
SGPT (U/L)	0.687	0.998 (0.990 – 1.007)		
SGOT (U/L)	0.431	1.002 (0.998 – 1.006)		
Door to balloon (min)	0.894	1.002 (0.973 – 1.032)		

*OR: Odd`s ratio, C.I: Confidence interval, LL: Lower limit, UL: Upper Limit, #: All variables with p<0.05 was included in the multivariate, *: Statistically significant at p ≤ 0.05*

Also it was found in RENASICA III registry [19] that in-hospital mortality was significantly higher in STEMI than in NSTEMI/UA this was explained by increase usage of invasive methods of treatment as well as late presentation of some patients that led to rapid deterioration and death.

It was found that in-hospital re infarction was statistically significant higher in STEMI than NSTEMI/UA patients.

This is similar to data in RENASICA III registry [19] that showed higher incidence of re infarction in STEMI than NSTEMI/UA patients.

Also in study done by Sultana et al [17] there was significant increase in risk of in-hospital re infarction in STEMI than in NSTEMI/UA patients.

This may be explained by presence of multivessel disease in some patients or increase thrombotic burden as seen in coronary angiography of many patients or may be due to extracardiac factors as uncontrolled diabetes that may occurred in the form of diabetic ketoacidosis that creates hypercoagulable state.

In our study CHF showed no statistically significant difference between STEMI and NSTEMI/UA in agree with a study done by

Table 9. Univariate and multivariate logistic regression analysis for the variables affecting in hospital Stroke (n = 12/200) for total sample

	Univariate		#Multivariate	
	p	OR (95% C.I)	p	OR (95% C.I)
Sex/ Female	0.205	2.133 (0.661 – 6.890)		
Age	0.002*	1.091 (1.032 – 1.153)	0.015*	1.079 (1.015 – 1.147)
Smoking	0.192	0.440 (0.128 – 1.511)		
Dyslipidemia	0.167	0.433 (0.133 – 1.418)		
In hospital reinfection	0.002*	8.900 (2.287 – 34.634)	0.013*	6.809 (1.488 – 31.158)
NSTEMI	0.100	2.686 (0.828 – 8.710)		
Killip class (/≥2)	0.101	2.729 (0.821 – 9.065)		
Echo	0.035*	1.071 (1.005 – 1.142)	0.065	1.066 (0.996 – 1.140)
Systolic blood pressure (mmHg)	0.111	1.015 (0.997 – 1.035)		
Diastolic blood pressure (mmHg)	0.368	1.017 (0.981 – 1.054)		
Pulse (b/pm)	0.320	1.018 (0.983 – 1.053)		
Fasting blood glucose (mg/dl)	0.077	0.985 (0.969 – 1.002)		
2hrs post prandial blood sugar(mg/dl)	0.524	0.998 (0.991 – 1.005)		
Hemoglobin (mg/dl)	0.480	0.864 (0.576 – 1.296)		
WBCs (cell/cubic mm×10^3)	0.733	1.013 (0.942 – 1.089)		
Platelets (cell/cubic mm×10^3)	0.279	0.995 (0.985 – 1.004)		
HDL (mg/dl)	0.989	1.000 (0.945 – 1.057)		
LDL (mg/dl)	0.112	0.984 (0.964 – 1.004)		
Cholesterol (mg/dl)	0.725	0.998 (0.988 – 1.009)		
Triglycerides (mg/dl)	0.143	0.990 (0.977 – 1.003)		
Creatinine (mg/dl)	0.280	0.241 (0.018 – 3.186)		
CK-MB (IU/L)	0.465	0.998 (0.992 – 1.004)		
SGPT (U/L)	0.534	0.994 (0.976 – 1.013)		
SGOT (U/L)	0.786	0.999 (0.992 – 1.006)		
Door to balloon (min)	0.412	1.018 (0.975 – 1.064)		

*OR: Odd`s ratio, C.I: Confidence interval, LL: Lower limit, UL: Upper Limit, #: All variables with p<0.05 was included in the multivariate, *: Statistically significant at p ≤ 0.05*

Belguith et al [22]) that showed no statistically significant difference between STEMI and NSTEMI/UA regarding CHF.

In our study in-hospital stroke occurred in small proportion of patients and showed no statistically significant difference between STEMI and NSTEMI/UA patient this came in agree with study done by Wojtkowska I et al [23] that showed no statistically significant difference in risk of stroke between STEMI and NSTMI/UA patients.

Also study done by Yaghi et al. [24] NSTEMI conferred a similarly increased risk of ischemic stroke as STEMI patients. This may be due to hypokinesia of myocardium which in-turn may cause formation of LV thrombus that may detach and reach any of cerebral arteries leading to ischemic stroke, also presence of malignant arrhythmia post ACS may have a role or may be due to presence of carotid plaques as a result of higher cholesterol and calcium level inside blood vessels causing ischemia.

The study had some potential limitations such as 1) small size of study population, as it's single center registry 2) many patients refused to do PCI due to cultural issues and many of them presented to us after 48 hours or more of symptoms only for assurance 3) Number of UA patients was small as our hospital is PCI capable center and most of referred cases were STEMI cases. Patients who received thrombolytic therapy and was successful or NSTEMI/UA patients refused to do PCI due to financial

issues. 4) We only reviewed in-hospital outcome and further research to discuss the long-term mortality and morbidity in details are needed.

5. CONCLUSION

Hypertension, diabetes and smoking are the major risk factors for ACS. In STEMI, most patients underwent PPCI that was reflected on small number of in-hospital mortality. Some patients received thrombolytic therapy and in most of them it was successful pharmacoinvasive and rescue PCI was done to successful and failed cases respectively. In patients presented with late presentation with no symptoms at time of examination conservative medical therapy was the treatment of choice. In Non-STEMI/UA patients risk stratification was done and so most of cases received conservative medical therapy and high-risk patients underwent early invasive PCI. In-hospital mortality was low that reflect the effectiveness of treatment.

CONSENT AND ETHICAL APPROVAL

As per university standard guideline participant consent and ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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