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Comparison between Non-obstructive versus obstructive coronary artery disease patients with acute coronary syndrome

Mohammed Salah El-den Abd Elsalam
Professor of cardiology, Faculty of Medicine - Al-Azhar University, Assuit, Egypt

Mahmoud Ahmed Abd Elbast
Lecturer of cardiology, Faculty of Medicine - Al-Azhar University, Assuit, Egypt

Ola Eliow Mohamed*
Cardiologist at the Ministry of Health in Sohag, Egypt
*Corresponding author

Ahmed El Tayeb
Lecturer of cardiology, Faculty of Medicine - Al-Azhar University, Assuit, Egypt

***Abstract*---**Background and aim: Differences in prognosis and baseline clinical presentation have been documented among patient with acute coronary syndrome and coronary artery disease with obstructive (ObCAD) or nonobstructive arteries (NObCAD), but the rates of events largely varied across single studies, the present study aimed to compare the clinical profile, in-hospital and 3-month outcomes major adverse cardiovascular events (MACEs) of Acute Coronary Syndrome (ACS) patients with Non – Obstructive Coronary Diseases (NOCAD), versus Obstructive Coronary Diseases (OCAD). Patients and methods: This prospective observational study included 200 consecutive patients admitted with the diagnosis of ACS to Coronary Care Unit (CCU) at Al- Azhar University hospital Assiut and Sohag Specialized Cardiac and Digestive System Center. Results: There was statistically significant increase in patients suffered from common complication of ACS during 3 months follow up in group B (CAD) than group A (NOCAD).There was statistically significant between number of vessels in both groups with heart failure, and recurrent angina pain complications. Conclusion: Female sex, young age, less diabetic patients, less smoking, less ST segment changes, elevations in peak troponin levels were all associated with coronary angiography showing no significant stenosis. Our data suggest that patients of NOCAD on treatment had a good prognosis in clinical

presentation after 3 Months and less in complications than CAD patients.

***Keywords*---**3 Non-obstructive, obstructive coronary artery disease, acute coronary syndrome.

Introduction

Acute coronary syndrome, the most severe manifestation of CAD, is burdened by a significant mortality, concerning approximately 5%–8% of the cases within six months of diagnosis **[1]** An important subset of ACS patients is reported to have either normal coronaries (NCs) or non-obstructive coronary artery disease (Non-CAD, defined as narrowing <50% lumen diameter) on angiography, with reported prevalence of (10%). **[2]**

The etiology and pathogenesis of CAD with angiographally normal coronary arteries are still a matter of debate .ACS without obstructive CAD (MINOCA) has been reported to be due to plaque disruption, plaque erosion, vasospasm, embolism, Spontaneous coronary dissection, and other causes. In addition, transient left ventricular dysfunction syndrome (takotsubo syndrome) is a form of MI without obstructive CAD and may be catecholamine toxicity, autonomic dysfunction, or a combination of these or other causes. Furthermore, myocarditis can present clinically as a syndrome meeting the universal definition of MI, with symptoms potentially attributable to ischemia, ECG changes, and biomarker elevation. [3]

Additional explanations result in symptoms that may be confused with ACS include, non-ischemic cardiac conditions including pericarditis, aortic dissection and non-cardiac conditions (pulmonary embolism, musculoskeletal causes, reflux esophagitis, esophageal spasm, gastritis or psychosomatic pain) [4] By Clinical history, ECG, cardiac enzymes, echocardiography, and coronary angiography, represent the first-level diagnostic investigations to identify the causes of MINOCA. Imaging modalities such as intravascular ultrasound, CT angiography, and MRI should also be considered to differentiate between plaque rupture, myocarditis, cardiomyopathy, and takotsubo syndrome **[5].** Compared with patients with obstructive CAD versus with non-obstructive CAD experienced lower rates of major cardiac events but remained at substantial risk and should be treated accordingly. **[6]**

A key branch point is ST-segment elevation (ST-elevation) or new left bundle-branch block on the electrocardiogram (ECG), which is an indication for immediate coronary angiography to determine if there is an indication for reperfusion therapy to open a likely completely occluded coronary artery. Separate Clinical Prarctace Guidelines (CPGs) have been developed for ST-elevation myocardial infarction (STEMI) **[7].** This work was aiming at observational study to compare the clinical profile, in-hospital and 3-month outcomes of major adverse cardiovascular events at the acute coronary syndrome patients with insignificant CAD and CAD. To noticed first 3- months complications of ACS with non-obstructive CAD and possible treatment and follow up.

Patients and Methods

This prospective observational study included 200 consecutive patients admitted with the diagnosis of ACS to Coronary Care Unit (CCU) at Al- Azhar University hospital Assiut and Sohag Specialized Cardiac and Digestive System Center.
The study was carried out over a period from October 2018 to august 2021 All patients with chest pain to be investigated and or acute coronary syndrome admitted in CCU , who will undergo coronary angiography and classified into 2 groups: Group A : Included 100 patients presented with ACS with angiographically normal or insignificant CAD (lumen diameter < 50%). Group B: Included 100 patients presented with ACS with angiographically significant CAD.

Non-obstructive CAD at angiography was defined as the absence of <50% stenosis in any major epicardial vessel. The 50% stenosis level has frequently been used as the definition of "significant" or "obstructive" CAD. Results were categorized as normal if all arterial segments were described as "normal" by the angiographer. Angiograms with segments described as having even minimal luminal irregularities were categorized as non-obstructive (or mild) CAD [8]. MINOCA was defined as the presence of a MI in the absence of obstructive coronary artery disease (ie, no epicardial vessel with a stenosis > 50% on angiography) [9].

Occlusion of the coronary artery which occurs at the site of an atherosclerotic plaque. Major adverse coronary events (MACE) were defined as death, reinfarction, recurrent angina, heart failure, cerebrovascular accident, and need for urgent procedure at 30 day. [2] Inclusion criteria: Patients included in our study are those patients presenting with ACS whether ST-segment or non ST-segment elevation Myocardial infarction. Exclusion criteria: Patients with contraindications or who did not undergo cardiac catheterization, advanced liver and kidney disease and previous CABG.

Methods

Each patient was subjected to:
Full history taking from all patients, including age, gender, smoking, hypertension ,diabetes mellitus ,dyslipidemia, and prior cardiac events: Systemic resting hypertension was defined as a resting systolic blood pressure >140 mmhg , a resting diastolic blood pressure >90 mmhg or chronic use of antihypertensive medications to lower blood pressure. Assessment of Diabetes Mellitus was based on patient history and medications used for treatment of D.M. Assessment of dyslipidemia were based on patient history of dyslipidemia, Smoking: Patients are classified into former or current smokers and non-smokers (including those who never smoked)

Clinical Examination: Full clinical examination was carried out for every patient with special emphasis on the following data: Heart rate & rhythm on admission. Blood pressure (BP) should be measured using a mercury sphygmomanometer and used an appropriately sized cuff (i.e. the ideal cuff should have a bladder length that is 80% and a width that is at least 40% of arm circumference, a length to width ratio of 2:1) was used. Head and neck examination: for arterial and

venous pulsation. Chest and heart examination: for heart sounds, additional heart sounds, murmurs and the back for lung congestion. Signs of congestive heart failure at presentation: Killip's classification: Killip class was evaluated at admission (Killip I, no heart failure; Killip II, S3 and/or basal lung crepitations; Killip III, acute pulmonary edema; Killip IV, cardiogenic shock). Upper and lower limb examination: for peripheral cyanosis and lower limb oedema.

Total laboratory investigations including

Cardiac enzyme measurement: Biomarkers (troponin I, CK-MB, or bedside troponin within the first 24 hours) done by VIDAS BIOMEIUX. Blood Sugar level: Patient is diagnosed to be diabetic when: Random plasma glucose ≥ 200mg dl. Lipid profile measuring was done by phenometer5010. Serum creatinine.

(Twelve-lead electrocardiogram (ECG)
(ECG) was done for all patients for ST segment Changes, arrhythmia, and reperfusion after PCI.

Trans-thoracic echocardiography

Transthoracic echocardiography performed with Simens Dimensions echocardiogram using 3, 5 MHz transducer. Assessment of the chambers size, the presence and degree of valvular heart disease, global and regional left ventricular systolic function and diastolic function were performed. The LV end-systolic and end-diastolic volumes were measured from the standard 2-dimensional apical 4- and 2-chamber projections using Simpson's biplane Method, and LV ejection fraction (LVEF) was calculated and M. Mode method. Echocardiography was done pre-discharge and follow up echocardiography was also done after 3 months.

In-hospital outcome: Mortality, development of heart failure / vs cardiogenic shock, and significant arrhythmia requiring treatment. Outcome after 3 month follow-up: Follow-up data were obtained by phone calls, or by periodic outpatient visits up to 3 months for the occurrence of MACE: Cardiac mortality, recurrent ACS, need for revascularization (PCI or CABG), heart failure and arrhythmia.

Coronary angiography (CA)
CA done by using Siemens Artis zee 20×20. Pre catheterization assessment: Full history, physical examination and pre cath lab including (Sr. creatinine – PT, PC, INR - and Serology) Contrast media: Xenetix 300mg/ml (Iobitridol), Maximum amount= 5(body weight)/Sr. Creatinine: Catheters: Judkins (J) & William (W) (JL 3.5 & JL 4, JR 3.5 & JR 4), Approaches: Percutaneous techniques usually from femoral artery were used. Projections:Selective left and right coronary angiograms performed in multiple angulated views: AP (anteroposterior) cranial &caudal. LAO (Left Anterior Oblique) cranial &caudal, RAO cranial &caudal and lateral view

Statistical analysis

Data collected throughout history, basic clinical examination, laboratory investigations and outcome measures coded, entered and analyzed using

Microsoft Excel software. The data collected were tabulated and analyzed by SPSS (statistical package for social science) version 25 (Armonk, NY: IBM Corp) on IBM compatible computer.

Results

The present study included 200 patients presented with acute coronary syndrome admission in CCU, the patients were divided into 2 groups: Group A (100 patients) included patients with non-obstructive CAD (all lesions < 50% stenosis). Group B (100 patients) included patients with obstructive CAD one or more lesions > 60 % stenosis or left main coronary artery obstruction.

In group A females were 50(50%) female, while in group B the females were 33(33%). So, Female were statistically significant higher in group A than group B. (p=0.015). As regards age, in group A the age ranged from 32 to 70 years with mean ± SD 51.40± 9.93, while in group B, the age ranged from 40 to 73 years with mean ± SD 58.00 ± 7.819. Table (1)

Cardiac enzyme troponin was Low peak in 5 (5 .0%) patients in group A, and 4 (4.0%) in group B, while in group A positive troponin in 95(95.0%) patients while in group B 96 (96.0%). Figure (1)

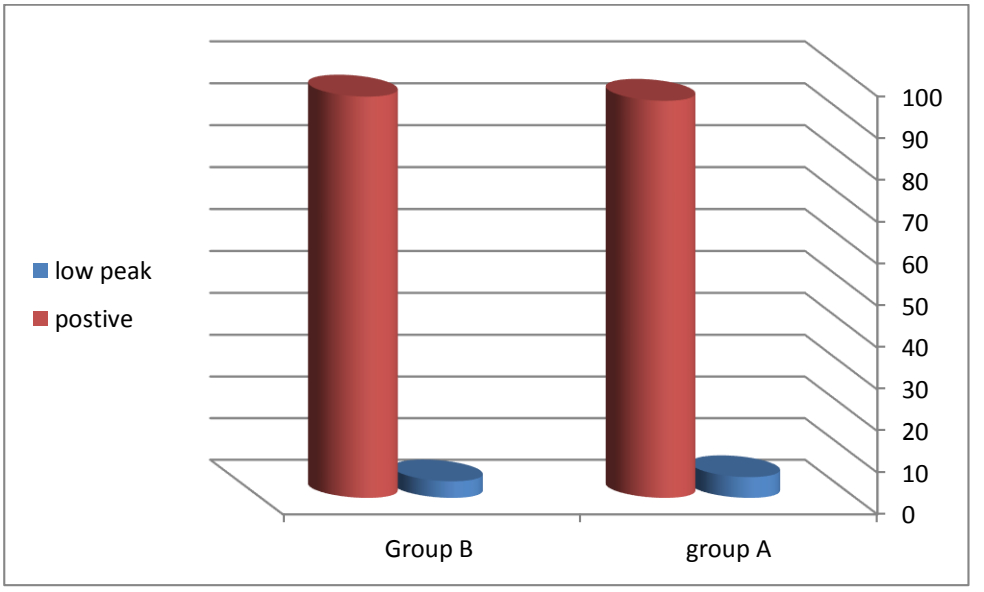

Figure (1): Comparison between group (A) and group (B) as regards Troponin

Angiogram results distribution: In group A was Atherosclerotic lesion < 50 patients 30 (30 .0%), Normal flow, 33(33.0%) Slow flow, 15 (15. 0%) Coronary spam 7 (7.0%) Ecstatic coronary 15 (15%). In group B was 9(9%) LCX, 50(50%) LAD, 19(19%) RCA, 3(3%) LM, LAD, 11(11%) LAD, LCX, 3(3%) RCA, LCX, and 5(5%) for CABG. Table (2)

Single vessel affected in NOCAD group 44 patients vs 58 patients of CAD group. Two vessels affected in NOCAD group 17 patients vs 30 patients of CAD group. Multi vessles affected in NOCAD group 6 patient's vs 12 patients of CAD group. 33 patients with normal flow in NOCAD group only. Table (3)

No statistically significant difference between the two groups at admission. Follow up ECHO after 3 months on treatment. Statistically significant improved LVEF in both groups after 3 months. And statistically significant improved patients of NOCAD group 78 % with presered LVEF than 52 % of patients of CAD group. Table (4)

This table shows that there was no statistically significant difference between the two groups as regards complications at admission. Table (5) There was statistically significant increase in patients suffered from common complication of ACS during 3 monthes follow up in group B (CAD) than group A (NOCAD). Table (6)

There was statistically significant between number of vessels in both groups with heart failure, and recurrent angina pain complications. There was no statistically significant between number of vessels in both groups with recurrent MI, arrhythmia, and death. NSTEMI patients were statistically significant higher in group NOCAD than group CAD while STEMI patients were statistically significant higher in group CAD than group NOCAD. no statistically significant between the two groups regrding cardiac enzyme troponin positive level. While, there was statistically significantly higher in group CAD than NOCAD group regards LDL level. No statistically significant difference between NOCAD vs CAD groups at admission in LVEF %. Statistically significant improved LVEF in both groups after 3 months. Statistically significant improved higher in patients of NOCAD group 78% with preserved LVEF than 52 % patients of CAD group. Table (7)

Table (1)
Comparison between group (A) and group (B) as regards demographic Data:

	Group A (n = 100)		Group B (n = 100)		P-value
	No.	%	No.	%	
Gender Female	50	50.0%	33	33.0%	0.015*
Age (years) (Min. – Max.) Mean ± SD.	(32 –70) 51.40± 9.93		(40–73) 58.00 ± 7.819		0.000**

p: p value for comparing between the studied groups

Table (2)
Distribution of angiogram results in group (A) and group (B)

Group	Angiogram results	Frequency	Percent
Group A (n = 100)	Normal flow	33	33 .0%
	Atherosclerotic lesion < 50	30	30 .0%
	Slow flow	15	15.0%
	Coronary spam	7	7 .0%
	Ecstatic coronary	15	15.0%
Group B (n = 100)	LCX	9	9.0%
	LAD	50	50.0%
	RCA	19	19.0%
	LM, LAD	3	3.0%
	LAD, LCX	11	11.0%
	RCA, LCX	3	3.0%
	for CABG	5	5.0%

Table (3)
Association between NOCAD and CAD regarding number of vessels affected:

P	X^2	Group B (N=100)	Group A (N=100)	Angiogram results
0, 001**	44 , 937	--------	33	No coronary lesions
		58	44	Single vessel diseases (1VD)
		30	17	Two vessel diseases (2VD)
		12	6	Multi vessel diseases (MV)

Association between NOCAD and CAD regarding number of vessels affected which was statistically significantly higher in group B than group A.

Table (4)
Comparison between group (A) and group (B) as regards ECHO at admission and after 3 months:

	Group A (n = 100)		Group B (n = 100)		P-value
	No.	%	No.	%	
ECHO at admission					
Severely impaired LVEF≤ 30 %.	14	14.0%	6	6.0%	
Impaired LV EF 36 - 49%.	12	12.0%	24	24.0%	0.089
Borderline low EF 50 - 54%.	48	48.0%	34	34.0%	
Normal LV EF ≥ 55%.	26	26.0%	36	36.0%	
ECHO after 3 months					
Severely impaired LVEF ≤ 30 %.	2	2.0%	3	3.0%	
Impaired LV EF 36-49%.	8	8 .0%	21	21.0%	0.004*
Borderline low EF 50 - 54%.	12	12.0%	24	24.0%	
Normal LV EF ≥ 55%.	78	78.0%	52	52.0%	

P-value	0.000**	0.002*	

*P-value ≤0.05 is significant **P- value ≤0.001 highly significant.

Table (5)
Comparison between group (A) and group (B) as regards complications in hospital

	Group A (n = 100)		Group B (n = 100)		P-value
	No.	%	No.	%	
Heart failure					
No	92	92.0%	86	86.0%	0.390
Yes	8	8.0%	14	14.0%	
Recurrent angina pain					
No	93	93.0%	82	82.0%	0.088
Yes	7	7.0%	18	18.0%	
Recurrent MI					
No	100	100.0%	97	97.0%	0.316
Yes	0	0.0%	3	3.0%	
Death					
No	100	100.0%	98	98.0%	0.316
Yes	0	0.0%	2	2.0%	
Arrhythmia					
No	96	97.0%	90	98.0%	0.651
Yes	4	3.0%	10	2.0%	

Table (6)
Comparison between group (A) and group (B) as regards complication after 3 months

	Group A (n = 100)		Group B (n = 100)		P-value
	No.	%	No.	%	
Heart failure					
No	81	81.0%	73	73.0%	0.007*
Yes	19	19.0%	27	27.0%	
Recurrent angina pain					
No	88	88.0%	76	76.0%	0.027*
Yes	12	12.0%	24	24.0%	
Recurrent MI					
No	99	99.0%	93	93.0%	0.030*
Yes	1	1.0%	7	7.0%	
Death					
No	99	99.0%	94	94.0%	0.030*
Yes	1	1.0%	6	6.0%	
Arrhythmia					
No	89	89.0%	78	78.0%	0.036*
Yes	11	11.0%	22	22.0%	

Table (7)
Association between NOCAD and CAD regarding number of vessels with complications:

P	X^2	Group B (N =100)				Group A (N=100)				Coronary Lesions / Complcations
		MV	2VD	1VD	NC	MV	2VD	1VD	NC	
0,012*	10, 880	13	6	8	-----	3	6	5	5	**Heart failure**
0,036*	8, 571	12	8	4	-----	2	4	3	3	**Recurrent Angina pain**
0,180	3, 429	4	2	1	-----	------	------	1	------	**Recurrent MI**
0,180	4,895	7	7	8	-----	3	4	2	2	**Arrhythmia**
0,212	1,556	4	2	-------	-----	-----	1	-----	------	**Death**

Case 1 (NOCAD):

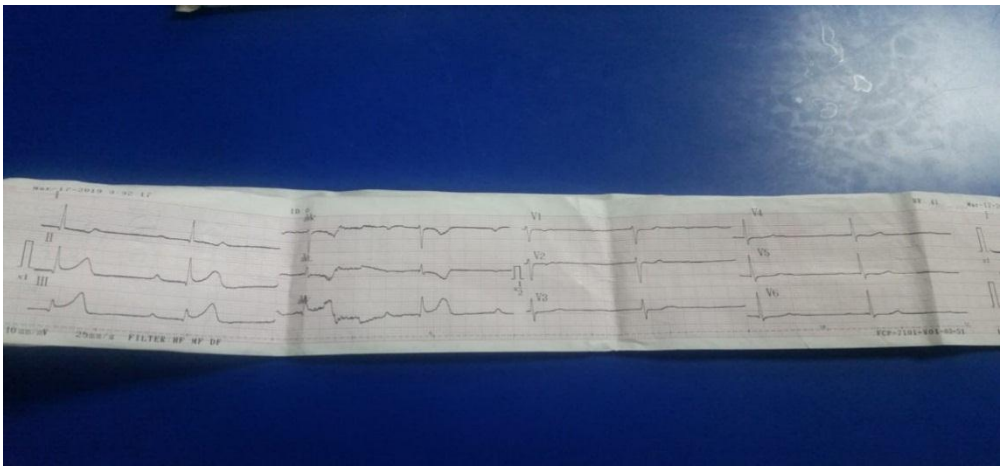

Figure (2): inferior STEMI

Figure (3): coronary normal flow

Case 2 (NOCAD):

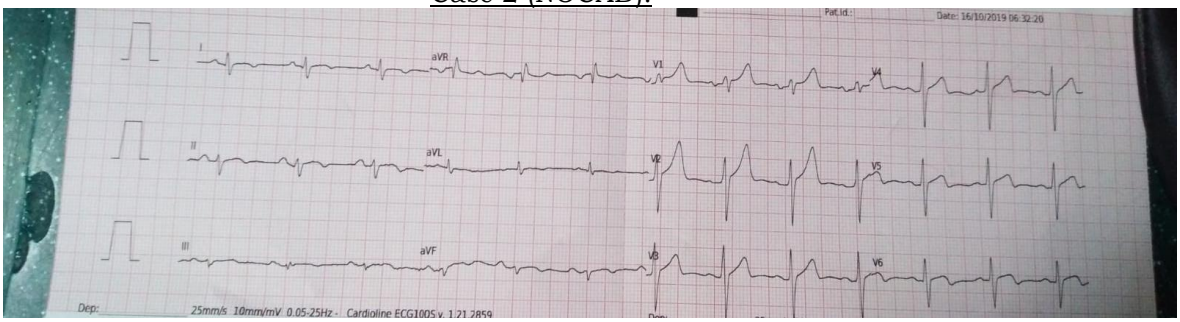

Figure (4): ECG inverted T wave in II, III , avl , and avf .

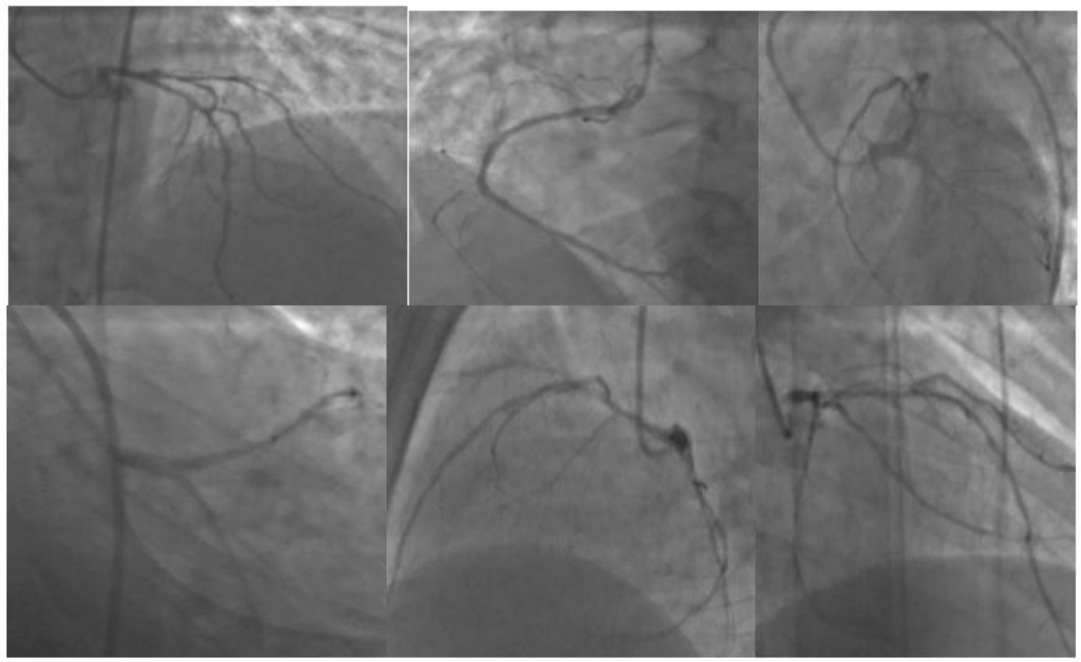

Figure (5): diffuse atherosclerosis <50% with small caliber of all coronaries

Discussion

MINOCA is a distinct type of myocardial infarction (MI) that was first described long ago; in a 2016 ESC position paper, it was considered a "working diagnosis" analogous to heart failure, prompting further evaluation regarding its underlying mechanisms. Ischemic heart disease (IHD) is a leading cause of death worldwide. Also referred to as coronary artery disease (CAD) and atherosclerotic cardiovascular disease (ACD), it manifests clinically as myocardial infarction. **[10].**
As regards age, in group A the age ranged from 32 to 70 years with mean ± SD 51.40± 9.93, while in group B, the age ranged from 40 to 73 years with mean ± SD 58.00 ± 7.819.

Patients with insignificant CAD (Group A) was significantly younger (< 35), than patients with significant CAD. (Group B). This was agree the study of Minha et al., [2] who analyzed the 2004-2010 ACSIS data, a total of 3,687 patients met the inclusion criteria. Of these, 84 patients had angiographic NC (2%), 79 patients had NOCAD (2%), who found that, the subset of patients without obstructive disease, compared to OCAD patients, tends to be younger ($p<0.001$). This result is similar to that in the study Berbesh et al., Patients with insignificant CAD were significantly younger (56 [50.0 – 61.5]) than CAD patients 65 [59.0 –71.0]) years **[11]**.

It is agreed with the study of Janosi Andras et al have reported The MINOCA pts were significantly younger (age 64.0 ± 14.4 vs. 65.5 ± 12.2 years) **[12]**.
In (group A) NOCAD 50(50%) females, while in (group B) than the females 33(33%) ($p=0.015$). In our study, patients with NOCAD were significantly more likely to be female gender, compared with patients with CAD patients.
This is agree with the study of Janosi Andras et al have reported the proportion of women was higher in NOCAD verses CAD. (55.7% vs 36.5) **[12]**.

Cardiac enzyme troponin was elevated in patients NOCAD (group A) , and 95 (95.0%) , and 96(96 .0%) in (group B) CAD patients, and there was no statistically significant difference between the two groups as regards cardiac enzyme troponin ($p=0.570$). 'Troponin -positive with non-obstructive coronary arteries (TpNOCA) The term TpNOCA refers to all conditions with elevated troponin levels (regardless of underlying etiology) plus non-obstructive coronary arteries on the coronary angiography, whereas MINOCA only refers to acute myocardial injury caused by myocardial ischemia **[13]**.

This result is similar to that in the study Berbesh et al., had elevations in peak troponin I (0.0005 [0 –0.74] vs. 53.5 [17.5–80.5], $p<0.001$) levels **[11]**. In group A, STEMI patients were 39(39.0%) and while in group B, STEMI patients were 53 (53.0%) and NSTEMI patients in group A were 61(61 .0%), while in group B , NSTEMI patients were 47(47 .0%). NSTEMI patients were statistically significant higher in group A than group B while STEMI patients were statistically significant higher in group B than group A. This result is similar to that in the study Ying et al, of the incidence of MINOCA reported in patients presenting with NSTEMI is about 8–10% and in STEMI cohorts it is 2.8–4.4%.22–25. **[14]**

This result is agree the study of Janosi Andras et al , the proportion of MINOCA patients among all MI was by 4.4% higher in of the STEMI group patients compared to the NSTEMI group (2.0% vs. 6.8%) **[12].** Among 100 patients with insignificant CAD (Group A), a normal coronary angiogram without any suspected atherosclerosis was present in (33%), whereas the rest (30 %) of patients showed signs of atherosclerosis (>0% and <50%), While slow flow patient 15% , and coronary spasm 7 %, ecstatic coronary 15 %. This is agree in the study of Yasser Yazied et al., [15] results 100 patients with insignificant CAD (Group I) a normal coronary angiogram (44%), and (56%) of patients showed signs of atherosclerosis (>0% and <50%).

All patients our study, as regards in hospital clinical outcome, 200 patients presented with acute coronary syndrome admission in CCU with typical chest pain and elevated in troponin level and all cases received full anti ischemic treatment , and anti-failure treatment and coronary angiography well done. No statistically significant difference between the two groups in ECHO results of LVEF at admission of CCU.

This was agree with the study of Berbesh et al , patients with insignificant CAD group had preserved left ventricular (LV) function pre-discharge, and even after discharge (p<0.0001) **[11]**. This was similar the result of the study of Minha et al., Most of the NONCA patients presented with normal or preserved left ventricular function on echocardiography, as compared with the significantly lower incidence of normal LV function in OCAD patients (77 vs. 45.5%, P<0.001). **[2]** There was statistically significant difference between the two groups as regards complications during 3 months follow up. Compare between both groups from complications in hospital, MINCAD vs. MICAD patients suffered from heart failure (9.3% vs. 11.1%), arrhythmias (10.8% vs. 10.6%), and history of Angina pectoris (7.8 % VS. 16.9%).

Overall, re-hospitalization rates were slightly lower over the 12-month follow-up period in patients with MINOCA compared with MICAD patients (38.2% vs. 41.1% P< 0.001), with MINOCA patients having fewer re-hospitalizations than MICAD patients for AMI or HF during 1st , 6th ,and 12th months follow up (re-hospitalizations AMI (0.3% , 0.8% and 1.3%) vs. (1.3% , 4.2% and 5.8%). That was agree with Larsen et al , at 3 years of follow-up, the patients with no-CAD versus CAD had lower rates of major adverse cardiovascular events (7.7% vs 22.2%, p = 0.002). **[16]**

And in the study of Carmine Pizzi et al., [17] In NObCAD and ObCAD patients, respectively, the combined yearly rates were as follows: 2.4% versus 10.1% (all-cause mortality); 1.2% versus 6.0% (myocardial infarction), 4.0% versus 12.8% (all-cause mortality plus myocardial infarction), 1.4% versus 5.9% (cardiac death), and 9.2% versus 16.8% (major cardiovascular events). In the studies directly comparing NObCAD versus ObCAD, all of the above outcomes were significantly less frequent in NObCAD subjects (with risk ratios ranging from 0.33 to 0.66). No differences in any outcome rate were observed between mild occlusion (1–49% stenosis) and zero occlusion patients.

In the result study of Minha et al., [2] In-hospital complications, including the incidence of heart failure, renal failure, and sustained ventricular arrhythmias, were comparable among the three groups, except for the transient ischemic attack rate, which was highest (1.3%) in the NOCAD group (*P*=.02). Complete follow-up was available for the majority of patients (100% of NC and NOCAD, and 99.6% of OCAD completed follow-up at 30 days , At 1 year, 78.6% of the NC group, 79.7% of the NOCAD group, and 74.6% of the OCSD group completed follow-up). At follow-up, patients with NC and NOCAD exhibited a significantly lower MACE rate at 30 days compared to patients from the OCAD group (4.8%, 3.8%, and 13.1%, respectively, P=*.02.* **[2]**

Results of the study of Ying et al., [14] MINOCA, irrespective of the underlying aetiology, is not a benign condition. A large systematic review and meta-analysis of 1,924 patients reported that all-cause mortality in unselected patients with MINOCA at 12 months was 4.7%. More recently, data from the 2003–2013 SWEDEHEART registry revealed that over a mean follow-up of 4 years, 23.9% patients with MINOCA experienced another major adverse cardiac event. In a retrospective registry of patients presenting with STEMI, those with MINOCA were reported to have a mortality rate of 3.6% at 30 days and 4.5% at 1 year.

This was not agree with Results of the study of Juarez et al., [18] about 12,814 individuals, 2,656 (20.7%) had normal coronary arteries, 2,254 (17.6%) had non obstructive CAD, and 7,904 (61.7%) had obstructive CAD. The risk of the primary outcome was increased in the non -obstructive group ($p = 0.01$) relative to those with normal coronary arteries. Non obstructive CAD was associated with an increased hazard of cardiovascular death and death of any cause ($p = 0.005$). There were no significant differences in the rate of acute myocardial infarction, stroke, or HF hospitalization.

Conclusion:

Female sex, young age, less diabetic patients , less smoking, less ST segment changes, elevations in peak troponin levels were all associated with coronary angiography showing no significant stenosis. Our data suggest that patients of NOCAD on treatment had a good prognosis in clinical presentation after 3 Months and less in complications than CAD patients

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