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Research Article

CLINICAL OUTCOMES IN ANGIOGRAPHIC DOCUMENTED CORONARY ARTERY DISEASE MANAGED WITH OPTIMAL MEDICAL THERAPY IN CURRENT ERA - ONE YEAR FOLLOW UP

Om Prakash[1], Vijay Pathak[1], Rashmi Gupta[2], Manish Ruhela[1]
[1]Department of Cardiology, SMS Medical College and hospital, Jaipur, Rajasthan, India
[2]Department of Preventive and Social Medicine, SMS Medical College and hospital, Jaipur, Rajasthan, India

Correspondence should be addressed to **Om Prakash**

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ABSRACT

Background

The management of patients with coronary artery disease (CAD) is complex. There is no study from India showing clinical outcomes in angiographically documented coronary artery disease in ACS and stable coronary artery disease in the current era. In most of previous studies only stable CAD patients were enrolled. Therefore the study is planned in view of above points to assess the outcome in patients with angiographically proven CAD on optimal medical therapy.

Methods

We conducted Hospital based observational descriptive prospective study involving 106 patients who had objective evidence of myocardial ischemia and significant coronary artery disease in at least one major epicardial vessel and not willing for either CABG or PCI, presenting to department of Cardiology, SMS Medical College from march 2013 to nov. 2013 and one year follow up was done till nov.2014. The clinical outcomes were recorded at six month and 12 months follow up.

Results

Most of the patients were male with a mean age of total patients' 58.93±11.49 years (range 32 to 83 years). Of these patients, 72 had diagnosis of previously stabilized ACS and 34 had stable coronary artery disease. The baseline characteristics of the patients were similar in the two groups. 45.28% of patients were symptomatic due to angina at 6 months. At 12 months, only 24.46% of patients had angina. There were no significant differences between the stabilized ACS group and non-ACS group in death (overall death 6.6%; 8.3% vs. 2.9%; P = 0.52); hospitalization for acute coronary syndrome (19.4% vs. 11.8%; P = 0.48); or myocardial infarction (overall MI 10.4%; 11.1% vs. 8.8%; P = 0.98), overall hospitalization rates (27.7%vs. 11.8% P=0.09).

Conclusions

Previously stabilized ACS as well as patients with stable coronary artery disease had similar outcomes with OMT so an initial management approach; optimal medical therapy can be implemented safely in the patients with previously Stabilized ACS as in patients with stable coronary artery disease.

KEY WORDS :ACS,Angiographically documented coronary artery disease,Non-ACSandOptimal medical therapy,

INTRODUCTION

Coronary artery disease (CAD) is the most important cause of mortality, but its age-standardized mortality has decreased by more than 40% during the last two decades[1,2]. Due to the decresed incidence in major risk factors and advance medical treatment technology.[3] Major prognostic factors of CAD were Advanced age, left-ventricular function, and the extent of coronary stenosis as assessed at the time of diagnostic coronary angiography and the presence or absence of more extensive atherosclerosis in other vascular beds.

Single-, double-, triple-, and left main CAD classification is also a major prognostic indicator during long-term follow-up of angiography studies[4][5].

The management of patients with coronary artery disease (CAD) is complex. In high-risk patients with acute coronary syndromes (ACS, with or without ST-segment elevation) a routine invasive strategy with revascularization in most patients provides the best outcome with a significant reduction in death and MI compared with an initial conservative strategy[6-9]. But similar benefit has not been shown in patients with stable coronary artery disease.[10][11][12]

As compared to medical therapy, percutaneous coronary interventions still carry a significant risk of acute periprocedural complications and follow-up reinterventions due to restenosis. The risk factor modification, like smoking cessation, exercise, diabetes mellitus management, lipid lowering, antianginal, and antihypertensive therapies play a major role in management of stable angina[13]. From advanced therapies like (PCI) and optimal medical therapy (OMT), it is not clear that which one carries better prognostic advantage, so further innovation in the field of pharmacologic and revascularization and PCI needed for betterment in the management of stable angina patients.

There is no study from India showing clinical outcomes in angiographically documented coronary artery disease in ACS and stable coronary artery disease in the current era. In most of previous studies only stable CAD patients were enrolled so no previous studies included both ACS and STABLE ANGINA patients. Several of previous trials were conducted in the time period before the availability of various newer antianginal drugs like Ivabradine[14-16]and Ranolazine[17-18] in clinical practice.

Therefore the study is planned in view of above points to assess the outcome in patients with angiographically proven CAD on optimal medical therapy.

METHODS

Study population

This Hospital based observational descriptive study was conducted at Cardiology department, SMS Hospital, Jaipur from march 2013 to nov.2014.patients were enrolled from march 2013 to nov 2013 and one year follow up was done till nov.2014. Entry criteria included patients having diagnosis of CSAP,UA,NSTEMI AND STEMI with 70% stenosis in at least one proximal epicardial coronary artery and objective evidence of myocardial ischemia (substantial changes in ST-segment depression or T-wave inversion on the resting electrocardiogram or inducible ischemia with either exercise or pharmacologic vasodilator stress) or at least one coronary stenosis of at least 80% and classic angina without provocative testing. Exclusion criteria included patients with previously documented PCI,previously diagnosed all heart failure patients(decompensated and compensated heart failure) ,known Hepatic and renal failure patients, patients with significant valvular heart disease, inability or unwillingness to consent, cross over from medical to PCI group during follow up. All eligible patients fulfilling inclusion criteria were explained about the nature and purpose of the study after taking their informed written consent. Clinical characteristics of these patients, baseline investigations, ECG, echocardiographic findings and treatment modality were recorded on a predescribedprorforma.All patients were assessed by use of a structured questionnaire regarding main risk factors and medical history. All patients were a detailed cardiac examination, and standard laboratory tests(HBA1C, CBC ,Blood urea , S Cretinine, B Sugar, Lipid profile)

Treatment

Allpatients received antiplatelet therapy with aspirin at a dose of 81 to 325 mg per day or 75 mg of clopidogrel per day, if aspirin intolerance was present. Diet, exercise and Medical antiischemic therapy included longactingmetoprolol, isosorbidemononitrate,ranolazine,nikorandil, trimetazidine and ivabradine alone or in combination with maximum tolerated doses, along with either remipril or losartan with maximum tolerated doses as standard secondary prevention. All patients received aggressive therapy to lower low-density lipoprotein (LDL) cholesterol levels according to guidelines. After achieving the LDL cholesterol target, next in order to increase high-density lipoprotein (HDL) cholesterol >40 mg per deciliter and lower triglyceride <150 mg per deciliter with the help of exercise, drugs (statins, extended-release niacin, or fibrates) alone or in combination. Blood pressure and blood sugar in diabetic patients were controlled according the guidelines.

Clinical outcomes

Clinical outcomes were recorded at six and 12 month follow up. Six month and one year follow-up were made by personal or telephone contact to these patients and patients were called for clinical reassessment and clinical events (if any) during follow up period. Ischemia was documented by either exercise stress testing or the typical stable angina assessment of the Canadian Cardiovascular Society (CCS).Outcomes were recorded at six month and one year follow up in terms ofclinical symptoms, like worsening angina with objective evidence resulting in hospitalization, acuteLVF,acute myocardial infarction,CABG and PCI during follow up, anddeath.

Statistical analysis:Sample size was calculated by assuming fifty four percent of patients with angiographically documented coronary artery disease having angina inspite of optimal medical therapy as per the result of previous studies, sample size required for present study was calculated to 82 patients at relative allowable error of 20% and 95% confidence interval.This was enhanced and rounded off to 90 patient assuming 10% attrition/drop out . So in this study we included 106Patients with angiographically documented lesions in at least one major epicardial vessel (defined as diameter stenosis >70%) with documented ischemia (substantial changes in ST-segment depression or T-wave inversion on the resting electrocardiogram or inducible ischemia with either exercise or pharmacologic vasodilator stress) or at least one coronary stenosis of at least 80% and classic angina without provocative testing and not willing for either CABG or PCI.

All data collected will be entered in excel sheet. Continuous variables were assessed by students T test.Categorical variables were assessed by chi –square test.Results were presented as mean and median with corresponding values (SD and inter-quartile ranges, respectively) and as percent.P value ≤ 0.05 was considered as significant. Outcomes were compared by dividing the patients in two groups as previously stabilized ACS(patients having UA,NSTEMI and STEMI previously with angiographically documented CAD) and non ACS group.

RESULTS

Baseline Characteristics and Angiographic Data

Between march 2013 and November 2014,we enrolled 106 patients with angiographically documented lesions in at least one major epicardial vessel (defined as diameter stenosis >70%) with documented ischemia. .Most of the patients were male with a mean age of total patients 58.93±11.49 years. Of these patients, 72 had diagnosis of ACS and 34 had stable coronary artery disease. Out of 72, 36 patients have STEMI,16 have NSTEMI and 20 have diagnosis of USAP. The baseline characteristics of the patients were similar in the two groups (Table 1). A total of 89 patients (83.9%) had objective evidence of myocardial ischemia, whereas in remaining 17 patients, exercise test was negative. 85% of the patients had multivessel coronary artery disease. LAD was involvement in around 86% of patients.STdepressions were found in 18 (16.9%) patients. T wave inversions were found in 63(59.4%) patients and Qwaves were found in 38 (35.8%) patients in resting ECG.In ECHO, 53(50%) patients had RWMA and 24(22.6%) patients had diastolic dysfunction.

Medication used during follow-up

Patients had a high rate of receiving multiple, evidence-based therapies during follow-up, in both study groups (Table 2).combination of Aspirin andclopidogrel was used only inpreviously stabilized ACS group. Clopidogrel was used in 5 patients of NON-ACS group as alternative to aspirin. Most commonly used antianginal drug was nitrates. Other antianginals likeNikorandil,Trimetazidine, Ranolazine and Ivabradine were used in 74.5%,46.2%,32% and 16% respectively. Most of the patients were taking two or more antianginal drugs durings follow up.

Clinical outcomes during follow up

angina status of patients was assessed according to the CCS classification during each visit.45.28% of patients were symptomatic due to angina at 6 months. At 12 months, only 24.46% of patients had angina. There was a substantial reduction in the prevalence of angina in both groups during follow-up. Statistically significant difference for angina was not found inpreviously stabilized ACS and NON-ACS groups throughout most of the follow-up period (Table 3). At 1 year, 83% of patients in NON-ACS group and 75% of those in thepreviously stabilized ACS group were free from angina. 6 patients were hospitalized due to acute heart failure,all these patients had previous ACS as compared to Non ACS group.(P=0.519NS) . Seven patients were hospitalized due to new episode of ACS. Overall hospitalization rate due to acute coronary syndromes and LVF was 27.7% in thepreviously stabilized ACS group and 11.8% in NON-ACS group on optimal medical-therapy (P=.48).Out of 106 patients, 7(6.6%) patients died during follow up(Table 3). In the NON-ACS group,3(8.8%) patients subsequently underwent CABG, as compared with 2(2.8%) patients in thepreviously stabilized ACS group. Revascularization was performed for angina that was unresponsive to maximal medical therapy or when there was objective evidence of worsening ischemia and having new episode of ACS. 11(10.4%) patients had STEMI during 1 year follow up. Out of these, death was occurred in 3 patients. The rates of myocardial infarction were 11.1% in thepreviously stabilized ACS group and 8.8% in NON-ACS group on optimal medical-therapy.

Subgroup analyses

Strong influences of syntax score and LM disease was found on clinical outcomes Meansyntax score was 22.34±12.64 with half of patients had syntax score of <22.Mean syntax score was comparable in bothpreviously stabilized ACS and NON-ACS group(p=.20).Clinical outcomes progressively increased across syntax score tertiles but difference was not statistically significant.LM disease had more adverse clinical outcomes in comparison to Multivessels and single vessels coronary artery disease

DISCUSSION

In our study it was found that all clinical outcomes at 1 year follow up showed no significant differences between the previously stabilized ACS and NON-ACS groups on optimal medical therapy because age, left-ventricular function, and the extent of coronary stenosis as assessed at the time of diagnostic coronary angiography and the presence or absence of more extensive atherosclerosis in other vascular beds are the most important prognostic factors in patients with established CAD and these factors were comparable in both the groups, but the rates of hospitalization and episodes of new acute coronary syndromes were more in previously stabilized ACS group in comparison to NON-ACS group(overall hospitalization rate 27.7% v/s 11.8% p=0.48). Ourfindings may be explained, in part, by differences in atherosclerotic plaque morphology and vascular remodelling associated with acute coronary syndromes, as compared with stable coronary artery disease. Vulnerable plaques (precursors of acute coronary syndromes) tend to have thin fibrous caps, large lipid cores, fewer smooth muscle cells, more macrophages, and less collagen, as compared with stable plaques, and are associated with outward (expansive) remodelling of the coronary-artery wall, causing less stenosis of the coronary lumen.[19] As a result, vulnerable plaques do not usually cause significant stenosis before rupture and the precipitation of an acute coronary syndrome.[19] By contrast, stable plaques tend to have thick fibrous caps, small lipid cores, more smooth muscle cells, fewer macrophages, and more collagen and are ultimately associated with inward (constrictive) remodelling that narrows the coronary lumen. These lesions produce ischemia and anginal symptoms and are easily detected by coronary angiography but are less likely to result in an acute coronary syndrome[20][21] Furthermore, our lower event rate than to COURAGE TRIAL[22]10.3% v/s12.3 %) with optimal medical-therapy may be explained by newer systemic therapy that reduced plaque vulnerability through aggressive intervention for multiple risk factors and evidence-based use of medication.

In our study, the rates of angina were similar in thepreviously stabilized ACS and NON-ACS group during follow-up, and rates of subsequent revascularization were likewise also similar. However, there was a substantial increase in freedom from angina at 6 month in comparison to RITA-2TRIAL[23](58% V/S 42%) and at 12 month in comparison to COURAGE TRIAL[22]76% v/s 67 %) with optimal medical therapy, most of which had taken place at 1 year but with a further improvement at 5 years. This finding reflects a benefit of specific antianginal medications (e.g., nitrates and beta blockers) or a favourable effect of therapies such as statins on endothelial function and atherosclerosis.

There was no significant difference in Mortality and myocardial infarction during 1 year follow in thepreviously stabilized ACS and NON-ACS group. The overall mortality rate was 6.6% of patients in our study which is comparable to COURAGE TRIAL[22]and reported in recent trials.[24-25]Similarily the rates of myocardial infarction were 10.3% which is also comparable to COURAGE TRIAL[22]and reported in recent trials.[24][25] These results are also concordant with a metaanalysis of all previous trials involving PCI versus medical management.[26]

In our study, Clinical outcomes progressively increased across syntax score tertiles but difference was not statistically significant. We studied the role of syntax score for prognosis in previously stabilized ACS and NON ACS patients treated with OMT but in SYNTAX trial[27] it was used in patients treated with PCI and CABG. These results of our study can make the SS an effective stratification tool when deciding the OMT in different syntax score tertiles in multivessels coronary artery disease.

Limitations of the study: The preponderance of male patients (83%) is a limitation of our study .One year follow up is very short compared to published trials. The number of patients in our study is very small compared to published trials so it is not large enough to detect small differences in outcomes. Our data is only from one center; hence our results are not generalizable. We enrolled both ACS and NON-ACS patients in study but in majority of previous trials, only NON-ACS patients were enrolled so no data available for comparisons.

CONCLUSION

Our findings reinforce existing clinical practice guidelines, which state that OMT can be safely used in patients with stable coronary artery disease, even in those with extensive, multi-vessel involvement and inducible ischemia, provided that intensive, multifaceted medical therapy is instituted and maintained. As our study results showed thatpreviously stabilized ACS as well as NON-ACS patients had similar outcomes with OMT so an initial management approaches, optimal medical therapy can be implemented safely inpreviously stabilized ACSpatients. Butthe results of this study need to be confirmed by further studies with larger populations.

REFERENCES

[1] Klenk J, Rapp K, Buchele G, Keil U, Weiland SK. Increasing life expectancy in Germany: quantitative contributions from changes in age- and disease-specific mortality. Eur J Public Health 2007;17:587–592.

[2] Lloyd-Jones D, Adams R, Carnethon M, De Simone G, Ferguson TB, Flegal K, Ford E, Furie K, Go A, Greenlund K, Haase N, Hailpern S, Ho M, Howard V, Kissela B, Kittner S, Lackland D, Lisabeth L, Marelli A, McDermott M, Meigs J, Mozaffarian D, Nichol G, O'Donnell C, Roger V, Rosamond W, Sacco R, Sorlie P, Stafford R, Steinberger J, Thom T, Wasserthiel-Smoller S, Wong N, Wylie-Rosett J, Hong Y. Heart disease and stroke statistics–2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2009;119:480–486.

[3] Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, Giles WH, Capewell S. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. N Engl J Med 2007;356:2388–2398.

[4] Emond M, Mock MB, Davis KB, Fisher LD, Holmes DR Jr, Chaitman BR, Kaiser GC, Alderman E, Killip T III. Long-term survival of medically treated patients in the Coronary Artery Surgery Study (CASS) Registry. Circulation 1994;90:2645–2657.

[5] Califf RM, Armstrong PW, Carver JR, D'Agostino RB, Strauss WE. 27th Bethesda Conference: matching the intensity of risk factor management with the hazard for coronary disease events. Task Force 5. Stratification of patients into high, medium and low risk subgroups for purposes of risk factor management. J Am CollCardiol 1996;27:1007–1019.

[6] Bassand JP, Hamm CW, Ardissino D, Boersma E, Budaj A, Fernandez-Aviles F, Fox KA, Hasdai D, Ohman EM, Wallentin L, Wijns W. Guidelines for the diagnosis and treatment of non-ST-segment elevation acute coronary syndromes. Eur Heart J 2007;28:1598–1660.

[7] Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE Jr, Chavey WE II, Fesmire FM, Hochman JS, Levin TN, Lincoff AM, Peterson ED, Theroux P, Wenger NK, Wright RS, Smith SC Jr, Jacobs AK, Halperin JL, Hunt SA, Krumholz HM, Kushner FG, Lytle BW, Nishimura R, Ornato JP, Page RL, Riegel B. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-Elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non-ST-Elevation Myocardial Infarction) developed in collaboration with the American College of Emergency Physicians, the Society for Cardiovascular Angiography and Interventions, and the Society of Thoracic Surgeons endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation and the Society for Academic Emergency Medicine. J Am CollCardiol2007;50:e1–e157.

[8] Van de Werf F, Bax J, Betriu A, Blomstrom-Lundqvist C, Crea F, Falk V, Filippatos G, Fox K, Huber K, Kastrati A, Rosengren A, Steg PG, Tubaro M, Verheugt F, Weidinger F, Weis M, Vahanian A, Camm J, De Caterina R, Dean V, Dickstein K, Funck-Brentano C, Hellemans I, Kristensen SD, McGregor K, Sechtem U, Silber S, Tendera M, Widimsky P, Zamorano JL, Aguirre FV, Al-Attar N, Alegria E, Andreotti F, Benzer W, Breithardt O, Danchin N, Di Mario C, Dudek D, Gulba D, Halvorsen S, Kaufmann P, Kornowski R, Lip GY, Rutten F. Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation: the Task Force on the Management of ST-Segment Elevation Acute Myocardial Infarction of the European Society of Cardiology. Eur Heart J 2008;29:2909–2945.

[9] Antman EM, Hand M, Armstrong PW, Bates ER, Green LA, Halasyamani LK, Hochman JS, Krumholz HM, Lamas GA, Mullany CJ, Pearle DL, Sloan MA, Smith SC Jr, Anbe DT, Kushner FG, Ornato JP, Jacobs AK, Adams CD, Anderson JL, Buller CE, Creager MA, Ettinger SM, Halperin JL, Hunt SA, Lytle BW, Nishimura R, Page RL, Riegel B, Tarkington LG, Yancy CW. 2007 Focused Update of the ACC/AHA 2004 Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines: developed in collaboration With the Canadian Cardiovascular Society endorsed by the American Academy of Family Physicians: 2007 Writing Group to Review New Evidence and Update the ACC/AHA 2004 Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction, Writing on Behalf of the 2004 Writing Committee. Circulation 2008;117: 296–329.

[10] Silber S, Albertsson P, Aviles FF, Camici PG, Colombo A, Hamm C, Jorgensen E, Marco J, Nordrehaug JE, Ruzyllo W, Urban P, Stone GW, Wijns W. Guidelines for percutaneous coronary interventions. The Task Force for Percutaneous Coronary Interventions of the European Society of Cardiology. Eur Heart J 2005;26:804–847.

[11] Hochman JS, Steg PG. Does preventive PCI work? N Engl J Med 2007;356: 1572–1574.

[12] Patel MR, Dehmer GJ, Hirshfeld JW, Smith PK, Spertus JA. ACCF/SCAI/STS/ AATS/AHA/ASNC 2009 Appropriateness Criteria for Coronary Revascularization: a report by the American College of Cardiology Foundation Appropriateness Criteria Task Force, Society for Cardiovascular Angiography and Interventions, Society of Thoracic Surgeons, American Association for Thoracic Surgery, American Heart Association, and the American Society of Nuclear Cardiology Endorsed by the American Society of Echocardiography, the Heart Failure Society of America, and the Society of Cardiovascular Computed Tomography. J Am CollCardiol 2009;53:530–553.

[13] Fraker TD, Jr, Fihn SD, Gibbons RJ, Abrams J, Chatterjee K, Daley J, Deedwania PC, Douglas JS, Ferguson TB, Jr, Fihn SD, Fraker TD, Jr, Gardin JM, O'Rourke RA, Pasternak RC, Williams SV, Smith SC, Jr, Jacobs AK, Adams CD, Anderson JL, Buller CE, Creager MA, Ettinger SM, Halperin JL, Hunt SA, Krumholz HM, Kushner FG, Lytle BW, Nishimura R, Page RL, Riegel B, Tarkington LG, Yancy CW; American College of Cardiology; American Heart Association; American College of Cardiology/ American Heart Association Task Force on Practice Guidelines Writing Group. 2007 chronic angina focused update of the ACC/AHA 2002 Guidelines for the management of patients with chronic stable angina: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines Writing Group to develop the focused update of the 2002 Guidelines for the management of

patients withchronic stable angina. *Circulation*. 2007;116:2762–2772.
[14] Borer JS, Fox K, Jaillon P, Lerebours G. Antianginal and antiischemic effects of ivabradine, an I(f) inhibitor, in stable angina: a randomized, double-blind, multicentered, placebo-controlled trial. Circulation 2003;107:817–823.
[15] Tardif JC, Ford I, Tendera M, Bourassa MG, Fox K. Efficacy of ivabradine, a new selective I(f) inhibitor, compared with atenolol in patients with chronic stable angina. Eur Heart J 2005;26:2529–2536.
[16] Ruzyllo W, Tendera M, Ford I, Fox KM. Antianginal efficacy and safety of ivabradine compared with amlodipine in patients with stable effort angina pectoris: a 3-month randomised, double-blind, multicentre, noninferiority trial. Drugs 2007;67:393–405.
[17] Nash DT, Nash SD. Ranolazine for chronic stable angina. Lancet 2008;372:1335–1341.
[18] Morrow DA, Scirica BM, Karwatowska-Prokopczuk E, Murphy SA, Budaj A, Varshavsky S, Wolff AA, Skene A, McCabe CH, Braunwald E. Effects of ranolazine on recurrent cardiovascular events in patients with non-ST-elevation acute coronary syndromes: the MERLIN-TIMI 36 randomized trial. JAMA 2007;297:1775–1783.
[19] Naghavi M, Libby P, Falk E, et al. From vulnerable plaque to vulnerable patient:a call for new definitions and risk assessment strategies. Circulation 2003;108:1664-72
[20] .Ambrose JA, Tannenbaum MA, Alexopoulos D, et al. Angiographic progression of coronary artery disease and the development of myocardial infarction. J Am CollCardiol 1988;12:56-62
[21] .Little WC, Constantinescu M, Applegate RJ, et al. Can coronary angiography predict the site of a subsequent myocardial infarction in patients with mild-tomoderate coronary artery disease? Circulation 1988;78:1157-66.
[22] Boden WE, O'Rourke RA, Teo KK, Hartigan PM, Maron DJ, Kostuk WJ, Knudtson M, Dada M, Casperson P, Harris CL, Chaitman BR, Shaw L, Gosselin G, Nawaz S, Title LM, Gau G, Blaustein AS, Booth DC, Bates ER, Spertus JA, Berman DS, Mancini GB, Weintraub WS; COURAGE Trial Research Group. Optimal medical therapy with or without PCI forstable coronary disease. *N Engl J Med*. 2007;356:1503–1516.
[23] RITA-2 Trial Investigators. Coronary angioplasty versus medical therapy for angina: the second Randomised Intervention Treatment of Angina(RITA-2) trial. *Lancet*. 1997;350:461–468.
[24] Hochman JS, Lamas GA, Buller CE, et al. Coronary intervention for persistent occlusion after myocardial infarction. N Engl J Med 2006;355:2395-407.
[25] De Winter RJ, Windhausen F, Cornel JH, et al. Early invasive versus selectively invasive management for acute coronary syndromes. N Engl J Med 2005;353:1095-104.
[26] Katritsis DG, Ioannidis JP. Percutaneous coronary intervention versus conservative therapy in nonacute coronary artery disease: a meta-analysis. Circulation 2005; 111:2906-12.
[27] Mohr FW, Morice MC, Kappetein AP, et al. Coronary artery bypass graft surgery versus percutaneous coronary intervention in patients with three-vessel disease and left main coronary disease: 5-year follow-up of the randomised, clinical SYNTAX trial. Lancet 2013;381:629–38

Table 1: Baseline Clinical and Angiographic Characteristics

	ACS(N=72)		*Non ACS(N=34)*		*Total*	*P Value LS*
Variables	*NO*	*%*	*NO*	*%*		
Female	*14*	*19.44*	*5*	*14.71*	*19*	*0.74 NS*
Male	*58*	*80.56*	*29*	*85.29*	*87*	
Diabetes	*15*	*20.83*	*14*	*41.18*	*29*	*0.05 NS*
Hypertension	*19*	*26.39*	*13*	*38.24*	*32*	*0.31 NS*
Smoker	*57*	*79.17*	*24*	*70.59*	*81*	*0.468NS*
Family History	*10*	*13.89*	*4*	*11.76*	*14*	*0.99 NS*
Dyslipidemia	*14*	*19.44*	*10*	*29.41*	*24*	*0.37 NS*
past history CVA	*5*	*6.94*	*4*	*11.76*	*9*	*0.64NS*
past history PAD	*6*	*8.33*	*2*	*5.88*	*8*	*0.95 NS*
ECHO						
RWMA	*44*	*61.11*	*9*	*26.47*	*53*	*0.002S*
Diastolic Dysfunction	*18*	*25.00*	*6*	*17.65*	*24*	*0.551 NS*
Syntax Score						
<22	*42*	*58.33*	*11*	*32.35*	*53*	*0.026 S*
>32	*12*	*16.67*	*13*	*38.24*	*25*	
22 to32	*18*	*25.00*	*10*	*29.41*	*28*	
Angiographic						

Diagnosis						
SVD	*14*	*19.44*	*2*	*5.88*	*16*	*0.126 NS*
DVD	*27*	*37.50*	*6*	*17.65*	*33*	*0.06NS*
TVD	*24*	*33.33*	*22*	*64.71*	*46*	*0.005S*
LM	*7*	*9.72*	*4*	*11.76*	*11*	*0.985NS*
ECG						
ST Depression	*12*	*16.67*	*6*	*17.65*	*18*	*o.879NS*
T Wave inversion	*53*	*73.61*	*10*	*29.41*	*63*	*<0.001 S*
Q Wave	*33*	*45.83*	*5*	*14.71*	*38*	*<0.004 S*
	Mean	*SD*	*Mean*	*SD*	*Mean±SD*	*P Value LS*
Positive Stress test	*59*	*81.94%*	*30*	*88.24%*		*0.58NS*
Age(Range 32 -83 yrs)	*57.78*	*11.55*	*61.38*	*11.15*	*58.93±11.49*	*.13*
LVEF	*48.33*	*11.23*	*46.44*	*11.33*	*47.73 ±11.24*	*.42*
Syntax score	*23.41*	*13.06*	*20.09*	*11.57*	*22.34 ±12.64*	*.20*

SVD-Single vessel disease, DVD- Double vessel disease, TVD-Triple vessel disease, LM- Left main disease, RWMA-Regional wall motion abnormality, LVEF- Left ventricular ejection fraction

Table 2:MEDICATIONS USED DURING FOLLOW UP

	TOTAL	ACS(72)		Non ACS(34)		
MEDICATION	No	NO	*%*	NO	*%*	P value
ASPIRIN	101	72	100	*29*	85.29	0.004 S
CLOPIDOGREL	69	64	88.89	5	14.71	<0.001S
STATIN	98	69	95.83	29	85.29	0.128NS
BETA BLOCKERS	104	71	98.61	33	97.06	0.82NS
NITRATES	105	71	98.61	34	100.00	0.7NS
ACE INHIBITORS/ARB	61	46	63.89	15	44.12	0.08NS
NIKORANDIL	79	46	63.89	33	97.06	<0.001S
IVABRADINE	17	11	15.28	6	17.64	0.979 NS
RANOLAZINE	34	21	29.17	13	38.24	0.477NS
TRIMETAZIDINE	49	29	40.28	20	58.82	0.114NS

Table 3:CLINICAL OUTCOMES DURING FOLLOW-UP

	ACS(72)		Non ACS(34)		Total	P Value LS
SYMPTOMATIC	*NO*	*%*	*NO*	*%*		
Baseline(N=106)	64	88.89	34	100.00	98	0.104 NS
First follow up(N=106)	30	41.67	18	52.94	48	0.75NS
Second Follow Up(N=94)	16	25.00	7	16.67	23	0.44NS
HOSPITALIZATION						
DUE TO ACS	14	19.4	4	11.8	7	0.48NS
DUE TO LVF	6	8.33	0		6	0.20NS
MI	8	11.11	3	8.82	11	0.98NS
Fatal MI	3	4.17	0	0.00	3	0.56NS
Non Fatal MI	5	6.94	3	8.82	8	0.95NS

CABG	2	2.78	3	8.82	5	0.37NS
DEATH	6	8.33	1	2.94	7	0.519NS
Non cardiac						
hepatitis B	1	1.39	0	0.00	1	0.7NS
Cardiac						
CVA	0	0.00	1	2.94	1	0.7NS
LVF	1	1.39	0	0.00	1	0.7NS
MI	3	4.17	0	0.00	3	0.56NS
VT	1	1.39	0	0.00	1	0.7NS

ACS- Acute coronary syndrome, LVF-Left ventricular failure, CVA-Cerebrovascular accident

Table 4:CLINICAL OUTCOMES DURING FOLLOW-UP ACCORDING TO SYNTAX SCORE

SYNTAX SCORE	Total	Death		CABG		MI	
	No	**No**	%	**No**	%	**No**	%
<22	53	2	3.77	2	3.77	3	5.66
22 to32	28	1	3.57	1	3.57	3	10.71
>32	25	4	16.00	2	8.00	5	20.0
Total	106	**7**	6.60	5	4.72	11	10.37
P Value LS		0.09 NS		0.675NS		0.153 NS	

Figure 1