



**PATTERN OF CORONARY ARTERY INVOLVEMENT IN PATIENTS WITH ACUTE CORONARY SYNDROME (ST SEGMENT ELEVATION MYOCARDIAL INFARCTION)**

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**ABSTRACT**

**Aims:** The aim was to study the pattern of coronary artery involvement in the patients presenting as Acute Coronary Syndrome - ST segment Elevation Myocardial Infarction (ACS-STEMI) in BPKIHS, Dharan. **Material and Methods:** This is a retrospective, descriptive study. A total of 50 consecutive patients of ACS-STEMI who underwent Percutaneous Coronary Intervention were analyzed for the pattern of coronary artery involvement at BPKIHS, Dharan. **Results:** Out of 50 patients of ACS (STEMI), anterior wall MI was present in 31 (62%), inferior wall MI was present in 19 (38%). SVD was present in 56%, DVD was present 28% and TVD was present in 16% of the patients with ACS (STEMI). Among 28 patients with SVD, LAD was the most common infarct related artery (IRA) in 18 patients (64.2%), RCA in 9 (32.1%) and LCX in 2 patients (7.14%). In patients with DVD, overall 78.5% had lesions in LAD and RCA, 14.3% had lesions in RCA and LCX and 7.14% had lesions in LAD and LCX. In patients anterior wall MI with TVD (6 patients- 19.35%), tubular to diffuse lesion with 80-100% occlusion of LAD, along with 70-80% stenosis of LCX and 50-90% stenosis of RCA were present. In 2 patients (10.52%) with inferior wall MI and TVD, there were total occlusion of RCA along with 70-80% stenosis of LAD and RCA. **Conclusion:** Anterior wall MI is more common than inferior wall MI. In patients presenting with anterior wall MI, single vessel disease is more common than DVD or triple vessel disease. LAD is the most common infarct related artery in anterior wall MI, and RCA is the most common infarct related artery in inferior wall MI. Total or subtotal thrombotic occlusion of Proximal LAD and Mid RCA are the most common lesions in patients with ACS (STEMI). Male gender, increased age, hypertension, diabetes mellitus, dyslipidemia, smoking are the well known risk factors of ACS (STEMI). In patients with diabetes mellitus DVD and TDV are more common than SVD.

**KEY WORDS:** ACS, STEMI, MI, CAG, LAD, LCX, RCA, SVD, DVD, TVD.

**INTRODUCTION**

With increase in incidence of risk factors like physical inactivity, overweight and obese states, dyslipidemia, hypertension, diabetes mellitus, tobacco use the incidence of Atherosclerotic coronary heart disease (CHD) is also increasing in both developed as well as in developing countries.<sup>[1,2,3,4]</sup> Atherosclerotic coronary heart disease may remain asymptomatic (subclinical disease), become obstructive (stable angina) and few may become vulnerable and lead to atherothrombotic events and manifest as acute coronary syndrome, sudden cardiac death or stroke.<sup>[1]</sup> On the one hand, atherosclerosis may diffusely involve the epicardial coronary arteries and even progress to total occlusion without causing clinically apparent episodes of instability or signs of infarction. On the other, sudden occlusion can cause infarction in the presence of very limited angiographically detectable atherosclerosis.<sup>[5]</sup> The pathogenesis of acute coronary syndrome begins with plaque rupture which activates the platelets and

coagulation cascade leading to thrombus formation. The thrombus leads to partial or complete coronary artery occlusion leading to various clinical manifestations of ACS.<sup>[6]</sup> Patients with acute coronary syndrome may present as ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI) or unstable angina (UA).<sup>[4,7]</sup>

Patients with STEMI typically have complete and persistent thrombotic occlusion of a large epicardial coronary artery with total cessation of coronary blood flow in the territory of the occluded epicardial coronary artery.<sup>[4,7]</sup> UA/NSTEMI usually results from the disruption of atherosclerotic plaque with a subsequent formation of platelet rich thrombus that obstructs coronary blood flow however there is no complete coronary occlusion.<sup>[7]</sup> Severity of coronary artery occlusion is responsible for the manifestation of ACS as STEMI or NSTEMI or UA.

In patients with STEMI besides starting appropriate medical management, prompt reperfusion of jeopardized myocardium is the most effective way of restoring the balance between myocardial oxygen supply and demand and limit the infarct size.<sup>[8,9]</sup> Reperfusion can be done by Fibrinolysis or by Mechanical reperfusion (Percutaneous coronary intervention-PCI or Coronary artery bypass graft-CABG).<sup>[10,11]</sup> PCI restores the normal blood flow (higher rates of thrombolysis in myocardial infarction (TIMI) flow grade 3 ) in the infarct-related artery more successfully than fibrinolysis.<sup>[9]</sup> The rate of reocclusions of the infarct-related artery resulting in reinfarction within subsequent months is less in PCI group as compared to fibrinolysis group.<sup>[9]</sup> Primary PCI is superior to fibrinolytic therapy in reducing short term mortality, nonfatal reinfarction, stroke. Diagnostic Coronary angiography (CAG) is a fundamental component of PCI. Identification of the coronary endoluminal lesion(s) responsible for ACS has become a central focus of both non- invasive and invasive treatment modalities in patients with coronary heart disease.<sup>[12]</sup> Since its introduction, conventional coronary angiography in the standard clinical means for depicting the coronary arteries and is “gold standard” for diagnosis CAD.<sup>[13]</sup> Focus on the culprit coronary lesion in patients with ACS allows recognition of the complex plaque and of presence of endoluminal thrombi that are closely associated to the mechanisms of the disease.<sup>[12]</sup> Both Qualitative (eye ball description of angiographic lesions) and Quantitative computer-based angiography (QCA) are important part of diagnostic coronary angiography. In our study we have included only qualitative analysis of the coronary lesions.

#### MATERIAL AND METHODS

This was a retrospective descriptive study carried out at Division of cardiology, Department of Internal Medicine,

BPKIHS, Dharan. The purpose of the study was to study the pattern of coronary artery involvement, severity of lesions in patients presenting as Acute Coronary syndrome ST segment Elevation Myocardial Infarction (ACS-STEMI). ACS-STEMI was diagnosed on the basis of sudden onset angina chest pain, new onset ST segment elevation (defined as new ST segment elevation of  $\geq 1$  mm in 2 or more consecutive chest leads or limb leads except  $V_2$ - $V_3$ , where the cut-off for ST elevation is  $\geq 2$  mm in men  $\geq 40$  years,  $\geq 2.5$  mm in men  $< 40$  years, or  $\geq 1.5$  mm in women),<sup>[14]</sup> positive cardiac enzymes. Patients of ACS-STEMI who presented within 12 hours of symptom onset, who present within 12 to 24 hours of symptom onset but having angina on presentation, or who present in cardiogenic shock, acute severe heart failure, were treated with Percutaneous Coronary Artery Intervention (PCI). Patients who presented late, who had renal impairment, who did not give consent for PCI were managed conservatively. Total of 50 consecutive patients of ACS-STEMI who underwent Percutaneous Coronary Artery Intervention (PCI) were enrolled for the study. Baseline clinical diagnosis, presence or absence of traditional risk factors for CAD (like smoking, hypertension, diabetes mellitus, dyslipidemia) were collected from hospital record and angiographic data were collected from cathlab database.

#### RESULTS

Our study included 50 patients presenting as Acute Coronary Syndrome, ST segment elevation myocardial infarction (ACS-STEMI). Our study population were in the wide age group ranging from 30-85 years. Male to female patients were in the ratio of 2.6:1. We tried to search the traditional risk factors like (hypertension, Diabetes Mellitus, Dyslipidemia, Smoking) among our study population. The baseline characteristics of our patients are defined in Table 1.

**Table 1: Baseline Clinical Characteristics of the study population.**

Baseline Clinical Characteristics (Total : 50 patients)	
Average age	60.58 $\pm$ 13.68 years
<u>Age Group</u>	
30-50 years	14 (28%)
51-70 years	25 (50%)
71-90 years	11 (22%)
<u>Gender</u>	
Male	36 (72%)
Female	14 (28%)
<u>Risk factors</u>	
Hypertension	31 (62%)
Diabetes Mellitus	19 (38%)
Smoking	28 (56%)
Dyslipidemia	22 (44%)

Depending upon ECG findings we diagnosed the patients as Anterior wall MI or Inferior wall MI. Most of our patients were having anterior wall MI (62%) compared to inferior wall MI (38%). Ischemic injury can produce conduction block at any level of the AV node or intraventricular conduction system and may present as

various grades of AV block, right or left bundle branch block, left anterior or left posterior (fascicular) divisional blocks. Disturbances of conduction can occur in various combinations.<sup>[15]</sup> Most of the patients had sinus rhythm, whereas complete heart block (CHB) was present in 2 patients, first degree AV block in 1 patient and atrial

fibrillation was present in 1 patient of inferior wall MI. Thirty three (.%) of the patients were in Killip I, 14 patients (%) were in Killip II, 1 patient was in class III and 2 (%) were in Killip IV.

In fifty percent of the study population coronary angiography (CAG) was done within 12 hours of chest

pain, in 23 patients (%) CAG was done between 12-24 hours because of persisting chest pain. One patient with inferior wall MI underwent CAG after 24 hours because of persistent CHB. CAG was done in one patient after 24 hours of chest pain due to cardiogenic shock. The clinical diagnosis of the patients are presented in Table 2.

**Table 2: Clinical presentation in patients with ACS (STEMI)**

Clinical Presentation in ACS (STEMI): Total 50 patients	
Anterior wall MI	31 (62%)
Inferior wall MI	19 (38%)
Killip I	33 (66%)
Killip II	14 (28%)
Killip III	1 (2%)
Killip IV	2 (4%)
<b>Rhythm</b>	
Sinus Rhythm	46 (92%)
Atrial Fibrillation	1 (2%)
First degree AV block	1 (2%)
Complete Heart Block	2 (4%)
<b>Window Period</b>	
<12 hours	25 (50%)
12-24 hours	23 (46%)
>24 hours	
Cardiogenic shock	1 (2%)
Complete Heart Block	1 (2%)
CAG post thrombolysis (Streptokinase)	2 (4%)

SVD was more common in both Anterior wall MI and Inferior wall MI (58% and 52.63%), followed by DVD (22.58% and 36.8%) and then TVD (19.35% and 10.52%). The angiographic profile of our patients is described in Table 3.

In patients with Anterior wall MI with SVD (18 patients), Left Anterior Descending Artery (LAD) was the culprit artery in all 18 patients (100%). Three (%) of them had complete thrombotic occlusion of ostial LAD, 5 patients (%) had total thrombotic occlusion of proximal LAD. Proximal LAD was subtotally occluded in 2 patients (%), 1 of the patient had recanalized proximal LAD. Mid LAD was subtotally occluded in 7 patients of Anterior wall MI. Seven patients (.%) of anterior wall MI had double vessel disease. Five of them had lesions in LAD and RCA, one had lesion in LAD and LCX, one had lesion in LCX and RCA. Among 5 patients with lesions in LAD and RCA, LAD was the culprit artery in all. In these patients there was total thrombotic occlusion of proximal LAD and mid LAD in each two of the patients, subtotal occlusion of mid LAD in one patient, whereas RCA was diffusely diseased in two patients and three had subtotal occlusion of mid RCA. In one patient of anterior wall MI with double vessel disease, there was subtotal thrombotic occlusion of proximal LAD with Chronic total occlusion (CTO) of proximal LCX. In one of the patient with anterior wall MI, proximal LCX was

subtotally occluded with significant diseases in proximal to mid RCA. Six (%) of our patients had triple vessel disease. In patients with TVD, tubular to diffuse lesion with 80-100% occlusion of LAD, along with 70-80% stenosis of LCX and 50-90% stenosis of RCA were present in 6 (19.35%) of the anterior wall MI patients.

Among patients presenting as inferior wall MI with SVD, 9 patients had RCA as the culprit artery, 1 patient had totally occluded LCX as the culprit artery. Out of these 9 patients with RCA lesions each 3 had total occlusion of proximal RCA and mid RCA, whereas in 2 patients there was subtotal thrombotic occlusion of mid RCA and in 1 patient distal RCA was totally occluded with heavy thrombus burden. Six (%) patients had lesions in RCA as the culprit artery along with the significant lesion in LAD. There was subtotal to total thrombotic occlusion of proximal RCA (2 patients), of mid RCA (3 patients) and total occlusion of PD in 1 patient. Each 3 of these DVD patients had 70-80% obstruction of proximal or mid LAD. In one patient of inferior wall MI, there was subtotal occlusion of proximal RCA along with diffuse significant stenosis of ostio-proximal LCX. Two patients of inferior wall MI had triple vessel disease in the form of total thrombotic occlusion of RCA along with tubular significant diseases in LAD and LCX.

**Table 3: Angiographic profile**

Angiographic profile						
Clinical Diagnosis	Artery Involved					
	SVD		DVD		TVD	
Anterior wall MI (31 patients)	<b>18 (58%)</b>		<b>7 (22.58%)</b>		<b>6 (19.35%)</b>	
	LM	0	LAD*+RCA	5	Culprit Artery   LAD	
	LAD	18	LCX*+LAD	1	LCX, RCA	
	LCX	0	LCX*+RCA	1		
	RCA	0				
Inferior wall MI (19 patients)	<b>10 (52.63%)</b>		<b>7 (36.8%)</b>		<b>2 (10.52%)</b>	
	LM	0	RCA*+LAD	6	Culprit Artery   RCA	
	LAD	0	RCA*+LCX	1	LAD, LCX	
	LCX	1	LCX*+LAD	0		
	RCA	9				

\* denotes culprit artery

We compared the pattern of coronary artery involvement in patients with ACS (STEMI) depending upon age, gender and presence or absence of risk factors like Hypertension, Diabetes mellitus, smoking. Table 4

shows the pattern of coronary artery lesions in these different groups of patients.

**Table 4: Pattern of coronary artery involvement in patients according to clinical profile.**

Clinical Profile	Artery involved in ACS (STEMI)			
	SVD	DVD	TVD	
Age group	30-50 years (14 patients)	<b>11 (78.5%)</b>	2 (14.3%)	1 (7.14%)
	51-70 years (25 patients)	<b>12 (48%)</b>	8 (32%)	5 (20%)
	71-90 years (11 patients)	<b>5 (45.6%)</b>	4 (36.7%)	2 (18.9%)
Gender	Male (36 patients)	<b>22 (61%)</b>	9 (25%)	5 (13.9%)
	Female (14 patients)	<b>6 (42.8%)</b>	5 (35.7%)	3 (21.4%)
Smoking	Smoker (28 patients)	<b>14 (50%)</b>	12 (42.8%)	2 (7.14%)
	Non smoker (22 patients)	<b>14 (63.6%)</b>	2 (9.09%)	6 (27.2%)
Diabetes Mellitus	Diabetic (19 patients)	5 (26%)	<b>8 (42%)</b>	6 (31.5%)
	Non diabetic (31 patients)	<b>23 (74%)</b>	6 (19.3%)	2 (6.45%)
Hypertension	Hypertensive (31 patients)	<b>16 (51.6%)</b>	9 (29%)	6 (19.3%)
	Non hypertensive (19 patients)	<b>12 (63%)</b>	5 (26%)	2 (10.5%)
Dyslipidemia	Dyslipidemic (22 patients)	<b>11 (50%)</b>	6 (27.7%)	5 (22.7%)
	Non dyslipidemic (28 patients)	<b>17 (60.7%)</b>	8 (28.5%)	3 (10.7%)

## DISCUSSION

The mean age of presentation in our study was 60.58±13.68 years, which was comparable to other studies done in India by Gupta R et al (57 ± 12 years),<sup>[16]</sup> in the western population as in COURAGE trial 62 ± 5 years conducted in USA, study by Hochman et al.<sup>[17]</sup> Fifty percent of our study population were in the age group of 51-70 years. Male patients were 36 (72%) in number and female patients were 14 (28%) in number, which was also a feature in study done by Rajni S et al.<sup>[16]</sup> This difference in male and female patients can be attributed to the gender bias and atypical presentation or could be that male gender itself is one of the risk factor for CAD. Hypertension was the most common risk factor among our patients (62%), followed by smoking (56%), dyslipidemia (44%) and diabetes mellitus (38%).

Anterior wall MI 31 patients (62%) was the most common type of MI in our study followed by Inferior wall MI in 19 (18%) of our patients. In our study population with acute STEMI (including both anterior

wall MI and Inferior wall MI), single vessel disease (SVD) was more common 28 patients (56%) than double vessel disease (DVD) (28%) or triple vessel disease (TVD)(16%). This is similar to the study done by Rajni S et al, where they had SVD 46.02%, DVD 15.94% and TVD (9.78%).<sup>[16]</sup> Similarly in a study done by Surender D et al SVD (56.6%) was the most common culprit artery in patients with ACS followed by DVD in 10.8% and TVD in 3.6%.<sup>[18]</sup> These findings are similar to the results in study done by Kumar et al<sup>[19]</sup> and Tewari et al.<sup>[20]</sup>

Among 28 patients with SVD, LAD was the most common infarct related artery (IRA) in 18 patients (64.2%), RCA in 9 (32.1%) and LCX in 2 patients (7.14%). In patients with DVD, overall 78.5% had lesions in LAD and RCA, 14.3% had lesions in RCA and LCX and 7.14% had lesions in LAD and LCX. Similarly in study done by Surender et al LAD was the most common artery involved (55.3%) followed by RCA lesions in 19% and then LCX in 11.8%.<sup>[18]</sup> Rajni S et al

in their study also have found LAD lesions more common followed by RCA and LCX lesions.<sup>[16]</sup> Lesions in proximal LAD was more common in all the patients with LAD as Infarct related artery whereas Mid RCA lesions were common than proximal or distal RCA lesions in patients with RCA as the culprit artery.

Single vessel disease was come common in all the age groups, however The prevalence of DVD and TVD increased with increasing age of the study population. SVD was also most commonly involved in male as well as female patients followed by DVD and TVD, similar to the studies done by Rajni S *et al.*,<sup>[16]</sup> Kumar *et al.*,<sup>[19]</sup> and Tewari *et al.*<sup>[22]</sup> However DVD and TVD were more common in female patients as compared to male patients. This may be due to atypical presentation of CAD in female patients. Diabetic patients had trend towards multi vessel disease in comparison to non diabetic subjects. We had 19 patients of diabetes mellitus in our study, 8 (42%) of them had DVD, 6 (31.5%) had TVD and 5 (26%) had SVD. Whereas in study done by Rajni S *et al.* 21.08% of the diabetic patients had DVD and 9.7% of the patients had TVD.<sup>[16]</sup> When considering pattern of involvement of coronary artery in hypertensive patients, SVD was the most common presentation in both hypertensive as well as non hypertensive patients. Also we noticed that DVD and TVD were more common in hypertensive patients as compared to non hypertensive patients. SVD was the common presentation in both smokers and non smokers, however we noticed that DVD was more common in smokers whereas TVD was more common in non smokers. In patients with dyslipidemia also SVD was more common than DVD or TVD. The prevalence of DVD was similar in patients with or without dyslipidemia, whereas TVD was more common in patients with dyslipidemia.

## CONCLUSION

Hence we concluded that Anterior wall MI is more common than Inferior wall MI. SVD is more common followed by DVD and TVD in both anterior and inferior wall MI. LAD is the culprit artery and total or subtotal occlusion of proximal is the most common lesion in patients with anterior wall MI. In patients with inferior wall MI, total or subtotal occlusion of the mid RCA is the most common lesion in our study. And we also concluded that SVD is the more common presentation of acute coronary syndrome in all the age groups, in both male and female gender and in patients with hypertension, dyslipidemia, history of smoking. Whereas in patients with diabetes mellitus multivessel disease (DVD, TVD) are more common than SVD.

## REFERENCES

1. Erling F, Valentin F. Atherothrombosis: disease burden, activity and vulnerability. In: Valentin F, Richard A, Robert A editors. *Hurst's The Heart*. 13<sup>th</sup> ed. USA:McGraw Hill, 2011.
2. Fuster V, Badimon L, Cohen M *et al.* Insights into the pathogenesis of acute ischemic syndromes. *Circulation* 1988; 77(6): 1213-1220.
3. Libby P *et al.* Current concepts of the pathogenesis of the acute coronary syndromes. *Circulation* 2001; 104(3): 365-372.
4. Kumar A, Christopher P *et al.* Acute Coronary Syndromes: Diagnosis and Management Part I 2009; 84(10): 917-938. Available from: Mayo Clin Proc.(2009).
5. Peter B, Stephen J, Sarah E. Comparison of Coronary Angiographic Findings in Acute and Chronic First Presentation of Ischemic Heart Disease. *Circulation* 1993; 87(6): 1938-1946.
6. Ahmed H, Adawey G, Essam M *et al.* Qualitative and Quantitative Coronary Angiography in patients with Acute Coronary Syndrome (ACS). *Egyptian Journal of Chest Diseases and Tuberculosis* 2012; 61: 209-215.
7. Robbert J, Jan GP *et al.* Non-ST-Segment Elevation Myocardial Infarction. Revascularization for Everyone?. *Journal of the American College of Cardiology* 2012; 5(9): 903-905.
8. Welch TD, Yang EH, Reeder GS. Modern management of acute myocardial infarction. *Current Probl Cardiol* 2012; 37: 237-248.
9. Albert S, Gjin N, Robert A. B, Adnan K. Percutaneous Coronary Intervention in Acute ST Segment Elevation Myocardial Infarction. In: Eric J Topol, Paul S.T editors. *Textbook of Interventional Cardiology*. 6<sup>th</sup> ed. California: Elsevier, 2012.
10. Jessica L M, David A M. ST-Elevation Myocardial Infarction: Management. In: Douglas LM, Robert OB, Douglas PZ editors. *Braunwald's Heart Disease A Textbook of Cardiovascular Medicine*. 10<sup>th</sup> ed. USA: Elsevier, 2015.
11. Jeffrey J. Popma. Coronary Arteriography. In : Douglas, LM, Robert OB, Douglas, PZ editors. *Braunwald's Heart Disease A Textbook of Cardiovascular Medicine*. 9<sup>th</sup> ed. USA:Elsevier, 2012.
12. E. Braunwald, E.M. Antman, Beasley. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non ST-segment elevation myocardial infarction. *Journal of American College of Cardiology* 2002; 40: 1366-1374.
13. E. Escolar, G. Weigold, A. Fuisz, N.J. Weissman. New imaging techniques for diagnosing coronary artery disease. *CMAJ* 2006; 174(4): 487-495.
14. Patrick TO, Frederick GK, Donal, E, James CF. ACCF/AHA 2013 Guideline for the Management of ST-Elevation Myocardial Infarction. *Journal of the American College of Cardiology* 2013; 61(4): 78-140.
15. Elliott M A, David A M. ST-Elevation Myocardial Infarction: Management. In: Douglas, LM, Robert OB, Douglas, PZ editors. *Braunwald's Heart Disease A Textbook of Cardiovascular Medicine*. 9<sup>th</sup> ed. USA: Elsevier, 2012.

16. Rajni S, Shivkumar B. Clinical characteristics, angiographic profile and in hospital mortality in acute coronary syndrome patients in south indian population. *Heart India* 2014; 2(3): 65-69.
17. Hochman JS, Tamis JE, Thompson TD, Weaver WD, White HD, Van de Werf F, et al. Sex, clinical presentation and outcome in patients with acute coronary syndromes. *Global Use of Strategies to Open Occluded Coronary Arteries in Acute Coronary Syndromes Iib Investigators. N Engl J Med* 1999; 341: 226-232.
18. Surender D, Tarun K, Rangaraj R, Chollenhalli N. M. et al. Demographic and angiographic profile in premature cases of acute coronary syndrome: analysis of 820 young patients from South India. *Cardiovascular Diagnosis and therapy* 2016; 6(3): 193-198.
19. Tewari S, Kumar S, Kapoor A, Singh U, Agarwal A, Bharti BB, et al. Premature coronary artery disease in North India: An angiography study of 1971 patients. *Indian Heart J* 2005; 57: 311-318.
20. Kumar N, Sharma S, Mohan B, Beri A, Aslam N, Sood N, et al. Clinical and angiographic profile of patients presenting with first acute myocardial infarction in a tertiary care center in Northern India. *Indian Heart J* 2008; 60: 210-214.