

Outcome of Thrombolytic Therapy in Hypertensives with Acute Myocardial Infarction

A. Prince Prabhakaran¹, Heber Anandan²

ABSTRACT

Introduction: Chronic Systemic Hypertension is one of the established cardiovascular risk factors for the development of atherosclerosis and increased incidence of peripheral vascular disease, chronic renal disease, and Coronary Artery disease.

Aim: To study the echocardiographic outcome of thrombolysis in hypertensive patients with AMI within 3 days and after 1 month of AMI and to detect left ventricular hypertrophy in such patients by electrocardiography and echocardiography and its influence on left ventricular systolic function.

Material and Methods: This is a prospective study to study the echocardiographic outcome of thrombolysis in the Hypertensive patient with Acute Myocardial infarction within 3 days and after one month and to detect left ventricular hypertrophy in such patient by ECG and Echo and its influence on left ventricular systolic function.

Results: Among the 26 cases with LVH diagnosed by Echo, 24 diagnosed having LVH with ECG with the sensitivity of 0.9 to 3. Among 24 cases diagnosed as No LVH by Echo, 21 cases were diagnosed as No LVH by ECG with a specificity of 0.875.

Conclusion: Patients with LVH after AMI had lower LVEF when compared to there without LVH. The Ejection fraction was shown to improve after one month irrespective of the presence of LVH. The most common conduction Abnormality in the study population was LBBB; Ventricular Tachycardia was commonly associated with Anterior wall MI with poor LVEF.

Keywords: Infarction, Thrombolysis, Coagulation, Hypertension

INTRODUCTION

CAD causes more deaths and disability and incurs greater economic costs than any other illness in the developed world and is on the rise in developing country.¹ The early (30 -day) mortality rate from AMI is 30%, with more than half of these deaths occurring before the stricken individual reaches the hospital.² Although the mortality rate after admission for AMI has declined by 30% over the past two decades, approximately 1 of every 25 patients who survive the initial hospitalization dies in the first year after AMI.³ Chronic systemic hypertension (HTN) is one of the established cardiovascular risk factors for the development of atherosclerosis and an increased incidence of peripheral vascular disease, cerebrovascular disease, chronic renal disease.⁴ Hypertension causes an increase in left ventricular mass and fibrous tissue resulting in increased stiffness of the left ventricle leading possibly to reduced coronary reserve, silent myocardial ischemia, and abnormal electrophysiological properties of hypertrophied

myocytes and conduction disturbances. Antecedent hypertension adversely affects mortality and heart failure after myocardial infarction. The presence of left ventricular hypertrophy is a significant indication of increased risk of adverse outcomes from ischemic heart disease.⁵ Myocardial infarction is a well-recognized cause which leads to heart failure with a preserved ejection fraction. Elevations of left ventricular end-diastolic pressure and ventricular volume and reduced ejection fraction are the most important signs of left ventricular dysfunction and are associated with a poor prognosis.⁶

Aim

To study the echocardiographic outcome of thrombolysis in hypertensive patients with AMI within 3 days and after 1 month of AMI and to detect left ventricular hypertrophy in such patients by electrocardiography and echocardiography and its influence on left ventricular systolic function.

MATERIAL AND METHODS

An observational prospective cohort study of patients receiving streptokinase for acute myocardial infarction. A total of 100 patients were included in the study. The patients were followed up at one month. A total of 100 cases of hypertensives with acute myocardial infarction admitted to ICCU of Tirunelveli Medical College Hospital, Tirunelveli. Only patients who survived the onset of MI and reached the ICCU where included in the surveys. The 30-day follow up was lost for three patients (6%), and they were excluded from the study.

Inclusion Criteria

1. Presence of typical chest pain suggestive of Acute myocardial infarction along with ECG evidence of Acute myocardial infarction who were thrombolysed. Criteria for thrombolysis being 2 mm or more ST elevation in two contiguous precordial leads or 1 mm or

¹Senior Assistant Professor, Department of Medicine, Tirunelveli Medical College and Hospital, ²Senior Clinical Scientist, Department of Clinical Research, Dr.Agarwal's Healthcare Limited, Tamilnadu, India

Corresponding author: A. Prince Prabhakaran, Senior Assistant Professor, Department of Medicine, Tirunelveli Medical College and Hospital, Tamilnadu, India

How to cite this article: A Prince Prabhakaran, Heber Anandan. Outcome of thrombolytic therapy in hypertensives with acute myocardial infarction. International Journal of Contemporary Medical Research 2018;5(5):E39-E43.

DOI: <http://dx.doi.org/10.21276/ijcmr.2018.5.5.27>

- more ST elevation in two contiguous limb leads. ECGs were recorded using BPL cardio art machine.
2. Time window of 12 hrs from the onset of pain to the initiation of thrombolysis.
 3. The patients have known hypertensives or those who presented with the blood pressure more than 140 systolic BP and > 90 diastolic BP.

Exclusion Criteria

1. Late thrombolysis (more than 12 hrs from the onset of pain)
2. Recurrent myocardial infarction.
3. Development of pericarditis.
4. Age < 18 and > 80 years.
5. Who died or lost in follow up in one month period.

All patients received streptokinase 1.5 million units in 100ms of Normal saline over 60 minutes. Aspirin was given to all patients. Use of heparin, β -blockers, ACE Inhibitors, Antihypertensives was according to ICCU protocols, which was by ACCAHA recommendations.

A detailed case history was taken for every patient who fulfilled the criteria for thrombolysis, and a meticulous examination was done as per proforma. A twelve-lead E.C.G. was recorded immediately after admission. Right precordial lead and posterior leads were also recorded. Patients were connected to the bed side cardiac monitor for 48 hours. E.C.G. was repeated subsequently each day, and additional E.C.G.s were taken as and when arrhythmias appeared. Complete 2D echocardiography examinations were performed using commercially available imaging systems (Aloka model SSD-830; 2.5 and 3.5-MHz transducers). Routine blood and urine investigations, serum cardiac enzymes, blood urea, serum creatinine, blood sugar, lipid profile, serum electrolytes and chest x-ray were done for all patients. 2D- Echocardiography was done on the 3rd day of admission. Evaluation of hemodynamic status was done daily by monitoring pulse, BP, JVP, cyanosis, urinary output and auscultation of heart and lungs. The average stay of patients in I.C.C.U. was 4 days. Their stay in I.C.C.U. was extended if any complications developed.

Statistical analysis was performed with the help of statistical

package SPSS (Statistical Package for the Social Sciences) version 11. Student t test were used to compare the data.

RESULTS

Maximum incidence of AMI occurred in the age group of 55-65 years (48%). Around 5(10%) patients were <45 years of age. The youngest male patient was 36 years, and the youngest female patient was 41 years. Majority of the study group were in the age group of 60-64. Out of 50 patients 38 (76%) of the cases were male. The male to female ratio was 3.16:1. Among all the patients, 52% of the cases presented with previous history of hypertension and rest presented with high blood pressure. Among 50 patients only 9(18%) were having DM diabetes mellitus. 71.1% males having the habit of smoking and 57.9% of them having alcoholism. Out of the 50 patients 33 (66%) of the cases having Dyslipidemia. Majority of patients (88%) admitted were wither in Killip's I / II Class. (Table 1) Anterior wall infarction was more common than inferior wall myocardial infarction. Right ventricle infarction is commonly associated with inferior wall myocardial infarction. Most of the patients 58% didn't have any arrhythmia. The most common arrhythmia was LBBB. The most common location of MI was Anterior wall, which may be accompanied by RWMA of septum and lateral wall. Among the 26 cases with LVH diagnosed by ECHO test, 24 having diagnosed as LVH by ECG with a sensitivity of 0.923. Among 24 cases diagnosed as no LVH by ECHO, 21 cases were diagnosed as no LVH by ECG with a specificity 0.875. About 34 patients had normal valves. Most common valvular abnormality was mitral regurgitation flowed by aortic regurgitation. (Table 2) After 1 month 37 patients had abnormal valves. Mean posterior wall thickness within 3 days is 1.44 mm and that after one month is 1.30 mm. The difference in wall thickness in these two observation is statistically significant (p, 0.05). Mean IV septal thickness within 3 days is 1.34 mm and that after one month is 1.23 mm. The difference in wall thickens in these two observation is statistically significant (p< 0.05). Mean LV internal diameter within 3 days is 3.63 mm and that after one month is 3.62 mm. This difference in wall thickens in these two observation is not statistically significant (p> 0.05). The mean LV ejection fraction was improved from 44.5% to 49.5% which is statistically significant (p< 0.05). (Table 3) The mean EF in patient with LVH was 41.8% while those without LVH was 47.8% in 3 days and the mean EF in patient with LVH was 50% while those without LVH was 48.2% after 1 month. But this difference was statistically insignificant. The change in EF at 3 days

KILLIP	Frequency	Percent
I	30	60.0
II	14	28.0
III	5	10.0
IV	1	2.0
Total	50	100.0

Table-1: The cardiac status according to Killip's class

Valves	Within 3 days		After 1 month	
	Frequency	Percent	Frequency	Present
Normal	34	68.0	37	74
Mitral regurgitation	11	22.0	8	16
Aortic regurgitation	4	8.0	3	6
Aortic stenosis	1	2.0	2	4
Total	50	100.0	50	100

Table-2: Presence of Valvular abnormality

Ejection Fraction	N	Mean	SD	t	p
Within 3 days	50	44.5	13.1	-5.347	0.0001
After 1 month	50	49.0	11.2		

Table-3: Mean ejection fraction

LVG-ECHO	N	EF within 3 days		t	p
		Mean	SD		
Absent	24	47.4	11.6	1.544	0.129
Present	26	41.8	14.1		

Table-4: Correlation of LVH with EF in 3 days

LVG-ECHO	N	EF After 30 days		t	p
		Mean	SD		
Absent	24	50.0	11.0	0.580	0.565
Present	26	48.2	11.5		

Table-5: Correlation of LVH with EF in 30 days

LVG-ECHO	N	EF After 30 days		t	p
		Mean	SD		
Absent	24	2.6	3.2	-2.339	0.024
Present	26	6.4	7.4		

Table-6: Correlation of LVH with change in EF in 30 days

Window period in hours	N	EF within 3 days		EF After 30 days		Change in EF	
		Mean	SD	Mean	SD	Mean	SD
<4	23	45.3	13.4	48.5	10.8	32	6.4
4-8	17	40.3	14.4	47.1	13.4	6.7	5.8
>8	10	49.7	8.0	53.7	6.7	4.0	4.6
Total	50	44.5	13.1	49.0	11.2	4.6	6.0
F		1.729		1.173		1.845	
P		0.189		0.318		0.169	

Table-7: Correlation of EF with Window period

and 30 days in patient with and without LVH was 6.4% and 2.6% respectively. (Table 4-6) The maximum number of patients (76%) were having stage II hypertension. 14% patients were having normal BP at the time of admission. Mean Ejection fraction of the cases with Normal BP was 37.3 within 3 days and changes to 41.0 after 1 month. Shock case has EF 26.0 within 3 days and becomes 48.0 after 30 days. Mean EF in different staffing was comparable in the observation made within 3 days and 30 days and also in the second observation. There was significant difference in the improvement in ejection fraction in different stages of BP. There was no significant correlation of EF with the duration of Window period. (Table 7) There was a significant improvement in EF after 1 month in patients with various arrhythmias and the change in EF was maximum in patients with VT. There was a significant difference ($p < 0.05$) in EF in patients with hypertension and diabetes mellitus and EF was low when compared to the patients with hypertension and diabetes mellitus.

DISCUSSION

In this study, it was observed that the mean EF in different stages of blood pressure was comparable in the observation made within 3-days and 30 days with ejection fraction

highest in normotensive. It was found to be low in patients with cardiogenic shock and those with stage 3 hypertension. Hypertension seems to play an especially important role in HF associated with a preserved ejection fraction (EF) >0.50 (HFPEF). No proven specific therapy exists for HFPEF, but the treatment of systolic hypertension in the aged (the group at highest risk for developing HFPEF) reduced the risk of developing HF by about one half.^{7,8}

Multivariate analysis by Lenzen MJ, Scholte op Reimer WJ, Boersma E, et al. showed that several of the predictors of death among patients with a preserved ejection fraction were related to those for patients with a decreased ejection fraction, as reported in previous studies.⁹

But in another study by Yusuf S, Pfeffer MA, Swedberg K, et al. in the Candesartan in Heart Failure: Evaluation of Reduction in Mortality and Morbidity (CHARM) trials observed a variation in mortality between patients with preserved ejection fraction and those with decreased ejection fraction¹⁰

First-degree AV block occurs in approximately 15% of patients who have an acute myocardial infarction (AMI), usually an inferior infarction. Almost all patients who develop first-degree AV block have conduction disturbances over the His bundle. In this study, the patients with first-

degree heart block had Inferior and Posterior wall MI. In 2% it was associated with right ventricular infarction also.

Mobitz type I, or Wenckebach, AV block occurs in almost 10% of patients who have an AMI and accounts for 90% of all patients who have an AMI and a second-degree AV block. A second-degree AV block is most commonly associated with an inferior MI. A Mobitz type II AV block accounts for 10% of all second-degree AV blocks (overall rate of <1% in the setting of AMI). It is most always associated with anterior infarction. This type of block often progresses suddenly to a complete heart block. In this study, none of the patients had Second degree AV block.

A third-degree AV block (ie., a complete heart block), occurs in 5-15% of patients who have an AMI and may occur with anterior or inferior infarctions. In this study only one patient had complete heart block, which was associated with inferior wall MI. In most massive patients, it settles within a few days without the need for a temporary or permanent pacemaker. The mortality rate for patients with inferior MI who develop complete heart block is about 15% unless a coexisting RV infarction is present, in which case the mortality rate is larger. The Cardiac Arrhythmias and Risk Stratification after Myocardial Infarction (CARISMA) trial monitored patients with acute myocardial infarction and decreased left ventricular ejection fraction and found that high-degree atrioventricular block was the most powerful predictor of cardiac death¹¹

In this study population only 6% of patients had LAHB and none had LPFB. Conduction from the His bundle is conveyed through 3 fascicles: the anterior division of the left bundle, the posterior division of the left bundle, and the right bundle. An abnormality of electrical conduction in 1 or more of these fascicles is noted in about 15% of patients with AMI. Isolated left anterior fascicular block (LAFB) occurs in 3-5% of patients with AMI; progression to complete AV block is uncommon. Isolated left posterior fascicular block occurs in only 1-2% of patients who have an AMI. The blood of supply of the posterior fascicle is larger than that of the anterior fascicle; therefore, a block here is associated with a relatively large infarct and high mortality rate.

In our study population, only 4% had RBBB, and 10% had LBBB. The patients with LBBB had poorer ejection fraction compared to those with RBBB in ECHO taken in 3 days. But the improvement in ejection fraction was better compared to those with RBBB. The right bundle branch receives its dominant blood supply from the left anterior descending (LAD) artery. Therefore, a new RBBB, which is seen in approximately 2% of patients with AMI, suggests a massive infarct territory. However, progression to complete heart block is uncommon. Mortality is frequently related to the amount of muscle loss. Bifascicular block in the presence of first-degree AV block is called a trifascicular block. In 40% of patients, a trifascicular block progresses to a complete heart block.

Around 8% of patient had VPCs. There not much reduction or improvement in ejection fraction after one month. In the past, frequent premature ventricular contractions (VPCs)

were considered to represent warning arrhythmias and indicators of impending malignant ventricular arrhythmias. However, presumed warning arrhythmias are frequently observed in patients who have an acute myocardial infarction (AMI) and who never develop ventricular fibrillation.

An accelerated idioventricular rhythm is seen in as many as 20% of patients who have an AMI.

RV involvement in inferior MI was associated with a higher risk of sustained VT and VF despite similar LV infarct size and function 58. Similarly, patients with residual RV dysfunction after inferior MI showed an increased risk of sudden death independent of LVEF⁵⁹. An alternative explanation may pertain to the difference in cardiac autonomic innervation.

In a study by Patrizio Pascale et al. observed that more than one-third of the patients with life-threatening ventricular arrhythmias in the chronic phase of MI have an LVEF \geq 40%. So LVEF is poor at distinguishing patients who will die from arrhythmia from those who will die of other cardiovascular cause.¹²

CONCLUSION

The hypertensive patients are prone for development of acute coronary syndrome due to accelerated atherogenesis. The chronicity of hypertension will cause left ventricular hypertrophy, within in turn lead to myocardial ischemia. Those patients with LVH, after AMI had lower LV ejection fraction when compared to those without LVH. The ejection fraction was shown to improve after one month irrespective of presence of LVH. The most common conduction abnormality in the study population was LBBB. Almost all patients with LBBB and LVH. Ventricular tachycardia was commonly associated with anterior wall MI with poor LVEF. But the above patients' LVEF was significantly higher in one month follow up.

REFERENCES

1. Kraitsoulas C, Anand SS. The impact of social determinants on cardiovascular disease. *The Canadian Journal of Cardiology*. 2010;26:8C-13C.
2. Elayalwar S, Natarajan S, Lakshmi pathi J. Serum neopterin: a new novel biomarker in acute myocardial infarction. *Journal of Evolution of Medical and Dental Sciences*. 2016;5:3582-3585.
3. Shivpuje A, Page S. Echocardiographic assessment of left ventricular function in patients of acute myocardial infarction. *International Journal of Advances in Medicine*. 2017;4:926.
4. Picariello C, Lazzeri C, Attanà P, Chiostrì M, Gensini G, Valente S. The Impact of Hypertension on Patients with Acute Coronary Syndromes. *International Journal of Hypertension*. 2011;2011:1-7.
5. Katholi R, Couri D. Left Ventricular Hypertrophy: Major Risk Factor in Patients with Hypertension: Update and Practical Clinical Applications. *International Journal of Hypertension*. 2011;2011:1-10.
6. Borlaug BA, Paulus WJ. Heart failure with preserved ejection fraction: pathophysiology, diagnosis, and treatment. *European Heart Journal*. 2011;32:670-679.
7. Kitzman DW, Gardin JM, Gottdiener J, et al.

- Importance of heart failure with preserved systolic function in patients \geq 65 years of age. *Am J Cardiol* 2001; 87:413-9.
8. Redfield MM, Jacobsen SJ, Burnett JC Jr, Mahoney DW, Bailey KR, Rodeheffer RJ. Burden of systolic and diastolic ventricular dysfunction in the community: appreciating the scope of the heart failure epidemic. *JAMA* 2003; 289: 194-202.
 9. Lenzen MJ, Scholte op Reimer WJ, Boersma E et al. Differences between patients with a preserved and a depressed left ventricular function: a report from the EuroHeart Failure Survey. *Eur Heart J* 2004; 25: 1214-20.
 10. Yusuf S, Pfeffer MA, Swedberg K, et al. Effects of candesartan in patients with chronic heart failure and preserved left ventricular ejection fraction: the CHARM preserved Trial. *Lancet* 2003; 362: 777-81.
 11. Bloch Thomsen PE, Jons C, Raatikainen MJ, MoerchJoergensen R, Hartikainen J, Viranen V, et al. Long-term recording of cardiac arrhythmias with an implantable cardiac monitor in patients with reduced ejection fraction after acute myocardial infarction: the Cardiac Arrhythmias and Risk Stratification After Acute Myocardial Infarction (CARISMA) study. *Circulation* 2010;122:1258-64.
 12. Patrizio Pascale et al Ventricular arrhythmia in coronary artery disease: limits of a risk stratification strategy based on the ejection fraction alone and impact of infarct localization, *EP Europace* Volume 11, Issue 12 PP. 1639-1646.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 01-05-2018; **Accepted:** 22-05-2018; **Published:** 13-06-2018