Ejection fraction and mortality rate of patients with isolated acute inferior myocardial infarction reperfused by streptokinase

Mozhdeh Beiraghdar[(1)], Zahra Pourmoghaddas[(2)], Mohammad Reza Torknezhad[(3)], Ali Torkan[(3)]

Abstract

BACKGROUND: This study aimed to evaluate the effects of streptokinase on left ventricular ejection fraction and mortality rate of patients with inferior acute myocardial infarction (AMI) without right ventricular myocardial infarction (RVMI).

METHODS: Fifty five consecutive patients with the diagnosis of inferior AMI without RVMI in the coronary care unit (CCU) of Shariati Hospital in Isfahan were selected for this study. Patients who had a history and/or electrocardiogram (ECG) evidence of previous myocardial infarction, evidence of bundle branch block, historical or clinical findings of valvular or other non-coronary heart diseases or heart failure were excluded. Participants were divided into two groups. Group one (n = 28) had no contraindication for taking thrombolytic therapy and group two (n = 27) had at least one contraindication for this treatment. Patients in group one took 1,000,000 units streptokinase for one hour. Three days later, LVEF of all participants was measured by an experienced cardiologist using 2-dimentional echocardiography. Patients were followed up until four weeks to assess the mortality rate.

RESULTS: One death in the first 24 hours was reported in group one. However, no death was reported in any group until four weeks after discharge. There was no significant difference in mortality rate during the first 24 hours and four weeks after discharge between the two groups. Mean LVEF in the two groups did not show any significant difference (P = 0.21).

CONCLUSION: Probably streptokinase has no effects on one-month mortality rate and LVEF in patients with inferior AMI without RVMI. Therefore, streptokinase side effects must be taken into consideration when being administered for this group of patients.

Keywords: Inferior Acute Myocardial Infarction, Left Ventricular Ejection Fraction, Streptokinase, Mortality Rate.

Introduction

Thrombolysis is claimed to be the treatment of choice for patients with acute myocardial infarction (AMI) less than 12 hours in duration who present ST segment elevations on their admission electrocardiogram (ECG), irrespective of the site of AMI.[1,2]

The risk to benefit ratio of thrombolytic therapy in patients with inferior AMI continues to be controversial.[3-6] It has been suggested that only patients with inferior AMI presenting larger ST segment deviations would benefit from thrombolytic therapy.[7] In addition, the effects of thrombolytic therapy on left ventricular ejection fraction (LVEF) have been small and inconsistent.[7-9]

Although hemorrhagic stroke is a rare and serious complication of thrombolytic therapy, if a patient has had a history of thrombolytic therapy before, an allergy against the thrombolytic agent may have developed. These two side effects of thrombolysis are worsened using streptokinase.[10-12]

In Iran, the most available thrombolytic agent used is streptokinase. Therefore, according to the side effects of this drug, we designed this study to assess streptokinase effects on LVEF and mortality rate in patients with inferior AMI without right ventricular myocardial infarction (RVMI).

Materials and Methods

This cross-sectional study was approved by the Ethics Committee of Islamic Azad University of Najaf Abad

1- MD, Assistant Researcher, Department of Cardiology, Najaf Abad Branch, Islamic Azad University, Isfahan, Iran.
2- MD, Assistant Researcher, Young Researchers Club, Najaf Abad Branch, Islamic Azad University, Isfahan, Iran.
3- Assistant Professor, Department of Cardiology, Najaf Abad Branch, Islamic Azad University, Isfahan, Iran.
Correspondence To: Zahra Pourmoghaddas, Email: zahrapormoghadas@yahoo.com

. In addition, all participants signed an informed consent.

Samples were selected from patients admitted in the coronary care unit (CCU) of Shariati Hospital in Isfahan, during September 23-March 19, 2008, diagnosed with inferior AMI without RVMI. Sampling method was convenient.

The diagnosis of inferior AMI without RVMI criteria included[9,13,14] chest pain or discomfort compatible with myocardial ischemia lasting longer than one hour, an elevation of 0.1 mV or more of the ST segment in three inferior leads (II, III, aVF) documented in the ECG within 4 hours of the onset of the symptoms, an elevation of serum creatinine phosphokinase (CPK) to 125% or more of the upper limit of the respective laboratory associated with the presence of MB-CPK, an ST segment elevation of 0.1 mV or more in the right precordial chest lead without V4R.

Patients were excluded if one of these items were present: historical or ECG evidence of previous myocardial infarction, evidence of bundle branch block, or historical or clinical findings of valvular heart disease or heart failure.

Finally, 55 patients were selected and divided into two groups. Patients in group one (n = 28) had no contraindication for thrombolytic therapy and patients in group two (n = 27) had at least one contraindication for taking thrombolytic agent.

The patients' weight and height were measured while they were barefoot wearing light indoor clothing. Body mass index (BMI) was then calculated for each subject as weight (kg) divided by height squared (m²).

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured twice with 5 minutes intervals using a standard mercury sphygmomanometer in a quiet and comfortable room.

Patients' blood samples were taken after 12 hours of fasting for measuring total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL-C), low density lipoprotein (LDL-C), and fasting blood sugar (FBS).

Patients were considered hypertensive if they had a history of high blood pressure and/or were using any anti-hypertensive drugs, and/or SBP ≥ 140 mmHg. Dyslipidemic patients were defined as having a history of dyslipidemia and/or TC ≥ 200 and/or TG ≥ 150 and/or LDL-C ≥ 100 and/or HDL-C ≤ 40 in males and ≤ 50 in females. Patients with FBS ≥ 126, and/or a history of diabetes mellitus, and/or taking anti-diabetic drugs were considered diabetic.

The contraindications for taking thrombolytic agent included a history of cerebrovascular hemorrhage at any time, a non-hemorrhagic stroke or another cerebrovascular event within the past year, SBP > 180 mmHg and/or DBP ≥ 110 mmHg at any time during the acute presentation, suspicion of aortic dissection, and active internal bleeding except for menses.[10]

Aspirin and intravenous heparin were administered to all patients. Other medications, including nitrates and calcium channel blockers, were used if considered necessary after the clinical examination by the physician. In addition, 1,000,000 units streptokinase during a one-hour period was ordered only for group one. An experienced cardiologist measured LVEF of patients using a 2-dimentional echocardiography 3 days after AMI.

In order to evaluate the mortality rate, the patients were followed up every week by phone until four weeks after discharge. Data were analyzed using SPSS version 13 and independent t-test was used to compare the two groups' data. P-value less than 0.05 was considered as statistically significant.

Results

As Table 1, including patients' physical and demographic data, shows, there were not any significant differences between the two groups.

Only one patient died in group one in the first 24 hours and no death was recorded during the 4 weeks after discharge in both groups. There was not a significant difference between the two groups in mortality rate at the first 24 hours (P = 0.51) and 4 weeks after discharge (P = 0.43).

Mean ejection fraction was 46.96 + 11.96% in group one and 51.11 + 12.58% in group two. Although ejection fraction in group one was 1.08 times more than group two, the difference was not significant (P = 0.21).

Discussion

Present study findings showed that mortality rate and LVEF of patients with acute inferior myocardial infarction without RVMI who took streptokinase is not higher than those who did not take this medication.

Despite more than a decade of extensive clinical trials, the use of thrombolytic treatment in patients with inferior wall AMI continues to be controversial.[1,4,12] Some studies have claimed that thrombolytic treatment should be given to all patients presenting an ST segment elevation.[13] Although the results are less convincing in patients with inferior AMI, treatment may save tens of thousands of lives worldwide each year.[1,2]

Borgia et al. concluded that thrombolytic therapy in patients with inferior AMI would improve the

Table 1. Demographic and physical characteristics of patients in the two groups

Data	Group one N = 28	Group two N = 27	P-value
Age (mean ± SD)	59.48 + 13.05	61.88 + 16.16	0.49
BMI (mean ± SD)	25.44 + 1.9	25.53 + 2.4	0.88
DM	39.1%	60.9%	0.09
HTN	45.8%	54.2%	0.30
DLP	63.2%	36.8%	0.14

BMI = body mass index; DM = diabetes mellitus; HTN = hypertension; DLP = dyslipidemia.

patients' prognosis (ST segment depression persisting for more than 24 hours) to a better prognosis (ST segment depression persisting for less than 24 hours).[12] Another study with the same aim showed that thrombolytic therapy in patients with inferior AMI presenting larger ST segment deviations is associated with improved 6-year survival rates.[10]

However, the risk to benefit ratio, in terms of early mortality in patients who have an ST of 0.smr or less no pericardial ST segment depression may be unfavorable.[9] In Iran, a study evaluating the role of thrombolytic therapy in inferior AMI with RVMI found LVEF in patients who received streptokinase decreased less than patient who did not receive streptokinase.[12]

In our study, there was no significant difference in ST segment depression in precordial leads between the two groups. Moreover, patients had no RVMI and their age, sex, history of diabetes mellitus, hypertension, dyslipidemia, smoking and addiction were matched. We found no significant differences in the mortality rate during the first 24 hours and one month after discharge between the two groups. This result is in agreement with a study conducted by Robalino, et al.[13] but our sample size was small. In addition, in this study ST segment depression was not significantly different between the two groups and we did not evaluate the effects of thrombolytic therapy on patients with RVMI.

Generally, this study shows that streptokinase has no effect on LVEF and mortality rate in the first 24 hours and one month after discharge. Considering that streptokinase is the only available thrombolytic agent in our country, the drug's side effects must be considered for administration in patients with inferior AMI without RVMI.

We suggest a study with a larger sample size for comparing the effects of streptokinase in patients with inferior AMI with or without RVMI.

Conflict of Interests

Authors have no conflict of interests.

References

1. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Lancet 1994; 343(8893): 311-22.
2. Randomized trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2.ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. J Am Coll Cardiol 1988; 12(6 Suppl A): 3A-13A.
3. Grines CL, DeMaria AN. Optimal utilization of thrombolytic therapy for acute myocardial infarction: concepts and controversies. J Am Coll Cardiol 1990; 16(1): 223-31.
4. Tobe TJ. Is thrombolytic therapy in acute inferior myocardial infarction really better than conventional treatment? Br Heart J 1995; 73(2): 108-9.
5. Zehender M, Kasper W, Kauder E, Geibel A, Schonthaler M, Olschewski M, et al. Eligibility for and benefit of thrombolytic therapy in inferior myocardial infarction: focus on the prognostic importance of right ventricular infarction. J Am Coll Cardiol 1994; 24(2): 362-9.
6. Schroder K, Wegscheider K, Neuhaus KL, Tebbe U, Schroder R. Significance of initial ST segment changes for thrombolytic treatment in first inferior myocardial infarction. Heart 1997; 77(6): 506-11.
7. Van de WF. Discrepancies between the effects of coronary reperfusion on survival and left ventricular function. Lancet 1989; 1(8651): 1367-9.
8. Meinertz T, Kasper W, Schumacher M, Just H. The German multicenter trial of anisoylated plasminogen streptokinase activator complex versus heparin for acute myocardial infarction. Am J Cardiol 1988; 62(7): 347-51.
9. Khani M, Taher Khani D. The survey of the effects of streptokinase on the patients suffering from inferiar wall infarction associated with right ventricular infarction. Journal Of Zanjan University Of Medical Sciences And Health Services 2001; 9(35): 4-9.
10. Further Readings American Heart Association. Heart and Stroke Facts: 2005 Statistical Supplement.

www.mui.ac.ir

Dallas: American Heart Association; 2006.

11. Murphy JF, Jain NL, Kahn MG. Inferiar wall myocardial infarction and thrombolytic therapy: an appraisal of clinical heterogencity and indications for treament. J Am cardial 1987; 59: 6-13.

12. Borgia MC, Gori F, Pellicelli A, Curcio D, Lionetti M, Buccarella PA, et al. Influence of thrombolytic therapy on inferior acute myocardial infarction with concomitant anterior ST segment depression.

Angiology 1999; 50(8): 619-28.

13. Robalino BD, Whitlow PL, Underwood DA, Salcedo EE. Electrocardiographic manifestations of right ventricular infarction. Am Heart J 1989; 118(1): 138-44.

14. Klein HO, Tordjman T, Ninio R, Sareli P, Oren V, Lang R, et al. The early recognition of right ventricular infarction: diagnostic accuracy of the electrocardiographic V4R lead. Circulation 1983; 67(3): 558-65.