International Journal of Research in Medical Sciences
Hirapur I et al. Int J Res Med Sci. 2018 Dec;6(12):3813-3818
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20184875

Original Research Article

Angiographic patency of streptokinase in STEMI patients: smokers vs. non-smokers

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Received: 29 August 2018
Revised: 22 October 2018
Accepted: 29 October 2018

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ABSTRACT

Background: Acute coronary syndrome is one of the leading causes of death. Smoking is known to be associated with many influencing factors for accelerating Myocardial Infarction (MI). In a country like India, Streptokinase (SK) is used as a leading therapeutic option for the treatment of ST elevation myocardial Infarction (STEMI). SK combines with plasminogen; this SK-plasminogen complex is responsible for fibrinolysis. The aim of this study was to determine angiographic patency after SK infusion in STEMI patients and comparison between smokers and non-smokers.
Methods: In this observational, prospective and single-centre study conducted between September 2011 and April 2012, a total of 398 patients who were diagnosed with STEMI were included. Patients were divided in two groups i.e. smokers and non-smokers. The patients were treated with thrombolytic (streptokinase) therapy and evaluated for TIMI 3 flow by performing angiography within 72hours of thrombolysis with SK.
Results: Of total 398 patients, 348 (87.4%) were male. The ratio of non-smokers and smokers was 1:2. Smokers were younger than the non-smokers (48.8±10.2 vs. 54.57±9.51). Post thrombolytic therapy, patients were evaluated for TIMI flow grades. Total of 202 patients achieved TIMI 3 flow, of which 157 were smokers and 45 were non-smokers.
Conclusions: Smokers have relatively hypercoagulable state than non-smokers. Better outcome in smokers group may be because of younger age and lesser comorbidities. Smokers should be motivated and guided properly to quit smoking.

Keywords: Thrombolytic therapy, Smokers, STEMI, Streptokinase

INTRODUCTION

Myocardial infarction (MI) is defined as the death of myocardium tissues due to reduced blood flow to the heart. Acute coronary syndrome (ACS) is one of the leading causes of death. Per year, more than one third of deaths in the developed countries are due to coronary heart diseases (CHD). Although, newer therapeutic approaches are available; MI still exists as one of the leading causes of death worldwide, affecting more than 7 million individuals each year.[1]

In 2010, estimated direct cost of hospitalisation because of MI was staggering 450 billion $ in US alone. Affluence in developing countries has also led to increase in CHD and MI.[2] Treatment options involve thrombolytic therapy and percutaneous coronary intervention (PCI).

Use of the thrombolytic therapy in patients suffering from acute MI was reported firstly by Fletcher and colleagues in 1958. In the next two decades, efficacy of intravenous streptokinase (SK) was evaluated by several trials.[3] Since then SK has become one of the most widely used thrombolytic agent for the management of MI. In country like India, SK is used as a leading therapeutic option for the treatment of MI.[4,5] Streptokinase is a 47 kDa protein (and not an enzyme), that combines with plasminogen; this SK-plasminogen complex is responsible for fibrinolysis.[6] Treatment with thrombolytic therapy can be evaluated with the coronary angiographic measurements of thrombolysis in myocardial infarction (TIMI) flow grades.

Ageing is one of the major influencing factors for manifestation of MI, followed by smoking. However, the harmful effects of smoking also increase with the age. Smoking falls under the list of one of the major preventable causes of MI.[7] Smoking is known to be associated with many influencing factors for accelerating MI. Despite having hazardous effects of smoking, many studies have reported that smokers get more advantageous effect of thrombolytic therapy than non-smokers for the treatment of MI; this is known as "the smoker's paradox", but no such study has been performed on Indian population.[8,11] Thus, the aim of this study was to determine angiographic patency after SK infusion in ST segment elevation myocardial infraction (STEMI) patients and comparison between smokers and non-smokers.

METHODS

Study design and population

This was an observational, prospective, single-centre study conducted between September 2011 and April 2012 at a tertiary care centre in India. A total of 398 patients were enrolled during this period. The patients who were admitted to the emergency department and diagnosed with STEMI were included in this study. STEMI was diagnosed in presence of two or three following criteria in patients: Chest pain and/or discomfort for at least 20minutes or more; ST segment elevation of >0.1 mm in at least two contiguous leads; in leads V_2-V_3 ST segment elevation of ≥0.2mm in men and ≥0.15mm in women in standard 12-lead electrocardiogram(ECG).[12,13]

The exclusion criteria were: (a) any prior intracranial haemorrhage (b) known structural cerebral vascular lesion (e.g., arteriovenous malformation) (c) known malignant intracranial neoplasm (primary or metastatic) (d) ischemic stroke within 3 months except acute ischemic stroke within 3 hours (e) suspected aortic dissection (f) active bleeding or bleeding diathesis [excluding menses] (g) significant closed head or facial trauma within 3 months (h) history of chronic severe poorly controlled hypertension (i) severe uncontrolled hypertension on presentation (SBP >180mmHg or DBP >110mmHg) (j) history of prior ischemic stroke >3 months, dementia, or known intracranial pathology not covered in contraindications (k) traumatic or prolonged (>10 min) CPR or major surgery (<3 weeks) (l) recent (within 2-4 weeks) internal bleeding (m) non-compressible vascular punctures sites (n) prior streptokinase, exposure (>5 days ago) or prior allergic reaction to these agents (o) pregnancy (p) active peptic ulcer (q) current use of anticoagulants: with high INR.

Study protocol

Basic characteristics like age, gender, blood pressure, heart rate, presence of diabetes or hypertension, smoking status and chest pain duration were determined at the time of admission for all the patients. According to their smoking status, the patients were divided into two groups, group I- "non-smokers" and group II- "smokers". The ratio was kept as 1:2 between the non-smokers and smokers, respectively. Blood samples for biochemistry analysis including lipid panel and ECG were also recorded. A written consent was received from each patient enrolled in the study.

Treatment approach

Intravenous infusion of 1.5million IU streptokinase was administered in all the STEMI patients. Infusion was given for a time of 1hour if there was no contraindication for thrombolytic therapy. Non-enteric 300mg aspirin and 300mg clopidogrel were given orally to all patients; other medications were prescribed but not limited to beta-blockers, angiotensin-converting enzyme inhibitors, low-molecular-weight heparin, IV nitrates and statins.

Angiographic study

Within 72hours of thrombolytic therapy, patients underwent coronary angiographic procedure using standard Judkins technique. Phillips machine was used to perform angiographic procedure. Coronary angiograms were interpreted by two experienced interventional cardiologists who worked independently and were unaware of clinical ailment and laboratory data of patients.

Study endpoints

After completion of thrombolytic therapy, angiographic TIMI flow rates were determined in the culprit vessel. Previously established grading system was used for the determination of TIMI flow rates, where TIMI 0: perfusion at the distal of stenosis, TIMI 1: penetration at the distal of stenosis but no perfusion; TIMI 2: partial perfusion, TIMI 3: complete perfusion.

Statistical analysis

The SPSS 15.0 software package (SPSS Inc., Chicago, IL, USA) was used for statistical analysis of the data.

Categorical variables were expressed as numbers (n) and percentages (%), whereas continuous variables were reported as mean and standard deviation or as median and interquartile range wherever appropriate. Chi-square test was used to compare categorical variables between the treatment groups. The Student's t-test or Mann-Whitney U test was used for comparisons between groups. The analysis of variance (ANOVA) or Kruskal-Wallis test was used for comparisons wherever applicable. The statistical level of significance for all tests was considered to be <0.05, confidence interval was 95%.

RESULTS

Baseline demographics

A total of 398 patients were included in this study during the mentioned study period. There were 129 patients in group I (non-smokers) and 269 patients in group II (smokers). Of the total population 348 (87.4%) were male and 265 of them were smokers. One hundred and thirty two (33.2%) patients were diabetic, and 67 of them were smokers.

Table 1: Baseline demographics and clinical characteristics of patients.

Characteristics	Total (N = 398)	Non-smoker (N = 129)	Smoker (N = 269)	P value
Demography				
Men, n (%)	348 (87.4%)	83 (64.3%)	265 (98.5%)	<0.001
Age (mean ± SD, years)	50.67 ± 10.35	54.57 ± 9.51	48.80 ± 10.23	<0.001
Diabetes, n (%)	132 (33.2%)	65 (50.4%)	67 (24.9%)	<0.001
Hypertension, n (%)	126 (31.7%)	58 (45.0%)	68 (25.3%)	<0.001
Family history, n (%)	69 (17.3%)	31 (24.0%)	38 (14.1%)	<0.05
Total Cholesterol (median (IQR), mg/dL)	184 (158 - 200)	184 (166 - 215)	180 (157-192)	<0.01
HDL (median (IQR), mg/dL)	38 (36-40)	38 (36-42)	38 (36-40)	0.097
TG (median (IQR), mg/dL)	171 (126-188)	175 (126-193)	166 (126-185)	0.527
LDL (median (IQR), mg/dL)	113 (95-121)	113 (97-129)	113 (95-118)	<0.05
Young age, n (%)				
0-40, years	79 (19.8%)	10 (7.8%)	69 (25.7%)	<0.001
>40, years	319 (80.2%)	119 (92.2%)	200 (74.3%)	
Window period, n (%)				
0-6 h	261 (65.6%)	76 (58.9%)	185 (68.8%)	0.074
7-12 h	109 (27.4%)	46 (35.7%)	63 (23.4%)	
13-18 h	10 (2.5%)	3 (2.3%)	7 (2.6%)	
19-24 h	18 (4.5%)	4 (3.1%)	14 (5.2%)	
Ejection fraction (EF), n (%)				
0-30, %	10 (2.5%)	3 (2.3%)	7 (2.6%)	0.422
31-45, %	210 (52.8%)	62 (48.1%)	148 (55.0%)	
45-60, %	162 (40.7%)	60 (46.5%)	102 (37.9%)	
>61, %	16 (4.0%)	4 (3.1%)	12 (4.5%)	
Coronary angiography timing, n (%)				
< 24 h	30 (7.5%)	11 (8.5%)	19 (7.1%)	0.138
≥24 h	112 (28.1%)	35 (27.1%)	77 (28.6%)	
≥48 h	163 (41.0%)	48 (37.2%)	115 (42.8%)	
≥72 h	22 (5.5%)	10 (7.8%)	12 (4.5%)	
≥96 h	40 (10.1%)	9 (7.0%)	31 (11.5%)	
≥120 h	23 (5.8%)	12 (9.3%)	11 (4.1%)	
≥144 h	8 (2.0%)	4 (3.1%)	4 (1.5%)	
Post SK ECG, n (%)				
Not settled	56 (14.1%)	11 (8.5%)	45 (16.7%)	<0.05
Settled	342 (85.9%)	118 (91.5%)	224 (83.3%)	
Next Day ECG, n (%)				
Not settled	16 (4.0%)	2 (1.6%)	14 (5.2%)	0.082
Settled	382 (96.0%)	127 (98.4%)	255 (94.8%)	
Thrombolysis in myocardial infarction (TIMI), n (%)				
TIMI 0	23 (5.8%)	9 (7.0%)	14 (5.2%)	<0.001
TIMI 1 & 2	173 (43.5%)	75 (58.1%)	98 (36.4%)	
TIMI 3	202 (50.8%)	45 (34.9%)	157 (58.4%)	

IQR: interquartile range; HDL: high density lipoprotein; LDL: low density lipoprotein; TG: triglycerides; SK: streptokinase; ECG: electrocardiogram

Table 2: Distribution of lesions among different segments of vessels.

Characteristics	Total (N = 398)	Non-smoker (N = 129)	Smoker (N = 269)	P value
Segments of LM, n (%)				
None	384 (96.5%)	121 (93.8%)	263 (97.8%)	0.064
Proximal	3 (0.8%)	3 (2.3%)	0 (0.0%)	
Distal	6 (1.5%)	3 (2.3%)	3 (1.1%)	
Diffuse	2 (0.5%)	1 (0.8%)	1 (0.4%)	
Ostial	3 (0.8%)	1 (0.8%)	2 (0.7%)	
Segments of LAD, n (%)				
None	111 (27.9%)	26 (20.2%)	85 (31.6%)	0.08
Ostio Proximal	50 (12.6%)	21 (16.3%)	29 (10.8%)	
Proximal	127 (31.9%)	42 (32.6%)	85 (31.6%)	
Mid	105 (26.4%)	37 (28.7%)	68 (25.3%)	
Distal	5 (1.3%)	3 (2.3%)	2 (0.7%)	
Segments of LCX, n (%)				
None	292 (73.4%)	87 (67.4%)	205 (76.2%)	<0.05
Ostio Proximal	30 (7.5%)	17 (13.2%)	13 (4.8%)	
Proximal	42 (10.6%)	17 (13.2%)	25 (9.3%)	
Mid	24 (6.0%)	6 (4.7%)	18 (6.7%)	
Distal	10 (2.5%)	2 (1.6%)	8 (3.0%)	
Segments of RCA, n (%)				
None	228 (57.3%)	67 (51.9%)	161 (59.9%)	0.202
Ostio Proximal	33 (8.3%)	12 (9.3%)	21 (7.8%)	
Proximal	54 (13.6%)	15 (11.6%)	39 (14.5%)	
Mid	65 (16.3%)	26 (20.2%)	39 (14.5%)	
Distal	18 (4.5%)	9 (7.0%)	9 (3.3%)	
Other Vessels 1, n (%)				
None	343 (86.2%)	115 (89.1%)	228 (84.8%)	0.552
Ramus	21 (5.3%)	6 (4.7%)	15 (5.6%)	
OM	2 (0.5%)	0 (0.0%)	2 (0.7%)	
Diagonals	32 (8.0%)	8 (6.2%)	24 (8.9%)	
Other Vessels 2, n (%)				
None	362 (91.0%)	117 (90.7%)	245 (91.1%)	0.874
Ramus	2 (0.5%)	1 (0.8%)	1 (0.4%)	
OM	32 (8.0%)	10 (7.8%)	22 (8.2%)	
Diagonals	2 (0.5%)	1 (0.8%)	1 (0.4%)	

LM: left main; LAD: left anterior descending; LCX: left circumflex; RCA: right coronary artery

There were seventy nine patients aged <40 years and suffered with STEMI, sixty nine of them were smokers. Smokers presented with the symptoms of STEMI at younger age than non-smokers and had significantly lower comorbid condition (p=0.001).

Other demographic details of patients including family history, window period, left ventricle ejection fraction, coronary angiographic timing, and post streptokinase ECG and TIMI flow details are presented in Table 1.

A total of 287 patients suffered from diseased left anterior descending artery. Further detailed information on lesions among different segments of vessels is presented in Table 2, and severity of lesion in vessels is depicted in Table 3.

Study outcomes

After completion of the thrombolytic therapy, the patients were evaluated for determination of TIMI flow rates. Of the total population, a total of 202 (50.8%) patients were able to achieve TIMI 3 flow. One hundred and fifty seven (58.4%) patients from group II (smokers) and 45 (34.9%) patients of the group I (non-smokers) achieved TIMI 3 flow. During the course of treatment some of the patient developed complications like contrast induced nephropathy, local site haematoma, post angiography pulmonary oedema, AV fistula etc. but all the complications were resolved later. Those patients who could not achieve TIMI 3 flow with thrombolytic therapy alone were managed medically and if condition got worse, aggressive medical management or PCI was considered, as indicated by concerned physician.

Table 3: Severity of lesions in different vessels.

Characteristics	Total (N = 398)	Non-smoker (N = 129)	Smoker (N = 269)	P value
Severity of LM, n (%)				
Normal	384 (96.5%)	121 (93.8%)	263 (97.8%)	<0.05
<50%	12 (3.0%)	8 (6.2%)	4 (1.5%)	
50-70%	1 (0.3%)	0 (0.0%)	1 (0.4%)	
>70%	1 (0.3%)	0 (0.0%)	1 (0.4%)	
Severity of LAD, n (%)				
Normal	111 (27.9%)	26 (20.2%)	85 (31.6%)	0.055
<50%	81 (20.4%)	24 (18.6%)	57 (21.2%)	
50-70%	33 (8.3%)	11 (8.5%)	22 (8.2%)	
>70%	173 (43.5%)	68 (52.7%)	105 (39.0%)	
Severity of LCX, n (%)				
Normal	292 (73.4%)	87 (67.4%)	205 (76.2%)	0.213
<50%	35 (8.8%)	15 (11.6%)	20 (7.4%)	
50-70%	27 (6.8%)	12 (9.3%)	15 (5.6%)	
>70%	44 (11.1%)	15 (11.6%)	29 (10.8%)	
Severity of RCA, n (%)				
Normal	228 (57.3%)	67 (51.9%)	161 (59.9%)	0.179
<50%	44 (11.1%)	12 (9.3%)	32 (11.9%)	
50-70%	26 (6.5%)	9 (7.0%)	17 (6.3%)	
>70%	100 (25.1%)	41 (31.8%)	59 (21.9%)	

LM: left main; LAD: left anterior descending; LCX: left circumflex; RCA: right coronary artery

DISCUSSION

Thrombolytic therapy prevents 20-30 deaths per 1000 patients with 25% reduction in mortality, as denoted by a review from fibrinolytic therapy trialist's group.[14] One of the landmark study for thrombolytic therapy; GUSTO-1 represented that, TIMI 3 flow after SK therapy was achieved only in 30% of patients, while in this study TIMI 3 was achieved in more than half (50.8%) of the patients.[15] Significantly higher number of patients from the smokers group achieved TIMI 3 flow than the non-smokers group ($p<0.001$), this could be because of the younger age of patients in smokers group (48.80±10.23 vs. 54.57±9.51, $p<0.001$) than the non-smokers. Along with age as an advantage, smokers were also less affected by other comorbidities like diabetes and hypertension. There was also significant difference in family history between smokers and non-smokers group (14.1% vs. 24.0%, $p<0.05$). Studies showing "the smoker's paradox" also showed similar data, where smokers were significantly younger than the non-smokers.[16-21]

Smoking accelerates atherosclerosis through various mechanisms and also has acute unfavourable effects on blood pressure and sympathetic tone. Along with atherosclerotic progression, long-term smoking may also accelerate oxidation of low-density lipoprotein and weaken endothelium-dependent coronary artery vasodilation. Smoking might also trigger spontaneous platelet aggregation, increase monocyte adhesion to endothelial cells, and adverse alteration in endothelial derived fibrinolytic and antithrombotic factors. Thus, the smokers are more susceptible to undergo such procedure earlier than the non-smokers and also smokers have less comorbidity at such young age than non-smokers. In a meta-analysis of 17 studies, only 6 demonstrated "the smoker's paradox" and concluded that; more focus should be on smoking cessation rather than relying on the "positive effects" of so called "the smoker's paradox".[22]

Smoking cessation is one of the most important interventions regarding cardiac morbidity and mortality. In a review, it was found that smoking cessation reduced coronary heart disease mortality by 36% when compared with mortality in patients who continued smoking.[23] The most important factors of any smoking cessation strategy include community education and physician-based primary prevention approach, also novel smoking cessation programme included direct financial assistance have already been evaluated and found effective.[24]

This study was limited to the measurement of the angiographic patency after thrombolytic therapy with SK. Follow-up of patients was not done. Patients who died during treatment or hospitalization were not included in this study.

CONCLUSION

Centres where provision for PCI is not available, thrombolytic therapy with SK can be a better substitute specifically in smokers and young patients. Smokers have

relatively hypercoagulable state than non-smokers. Better outcome in smokers group may be because of younger age and lesser comorbidities. Smokers should be motivated and guided properly to quit smoking.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Mendis S, Thygesen K, Kuulasmaa K, Giampaoli S, Mähönen M, Ngu Blackett K, et al. World Health Organization definition of myocardial infarction: 2008-09 revision. Int J Epidemiol. 2010;40(1):139-46.
2. Reed GW, Rossi JE, Cannon CP. Acute myocardial infarction. Lancet. 2017;389(10065):197-210.
3. White HD, Van de Werf FJ. Thrombolysis for acute myocardial infarction. Circ. 1998;97(16):1632-46.
4. White H. Selecting a thrombolytic agent. Cardiol Clin. 1995;13(3):347-54.
5. Banerjee A, Chisti Y, Banerjee U. Streptokinase-a clinically useful thrombolytic agent. Biotechnol Adv. 2004;22(4):287-307.
6. Ali MR, Salim Hossain M, Islam MA, Saiful Islam Arman M, Sarwar Raju G, Dasgupta P, et al. Aspect of thrombolytic therapy: a review. Scientific World J. 2014;2014:8.
7. Grundy SM, Pasternak R, Greenland P, Smith S, Fuster V. Assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American heart association and the American college of cardiology. Circ. 1999;100(13):1481-92.
8. Jaatun HJ, Sutradhar SC, Dickstein K. Comparison of mortality rates after acute myocardial infarction in smokers versus non-smokers. Am J Cardiol. 2004;94(5):632-6.
9. Suriñach J, Alvarez L, Coll R, Carmona J, Sanclemente C, Aguilar E, et al. Differences in cardiovascular mortality in smokers, past-smokers and non-smokers: findings from the FRENA registry. Eur J Intern Med. 2009;20(5):522-6.
10. Gupta T, Kolte D, Khera S, Harikrishnan P, Mujib M, Aronow WS, et al. Smoker's paradox in patients with st-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. J Am Heart Assoc. 2016;5(4):e003370.
11. Chen KY, Rha SW, Li YJ, Jin Z, Minami Y, Park JY, et al. 'Smoker's paradox'in young patients with acute myocardial infarction. Clin Exp Pharmacol Physiol. 2012;39(7):630-5.
12. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR. ESC/ACCF/AHA/WHF expert consensus document. Circ. 2012;126(16):2020-35.
13. Nikus K, Birnbaum Y, Eskola M, Sclarovsky S, Zhong-qun Z, Pahlm O. Updated electrocardiographic classification of acute coronary syndromes. Curr Cardiol Review. 2014;10(3):229-36.
14. Trialists FT. 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. Lancet. 1994;343(8893):311-22.
15. Investigators G. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. N Engl J Med. 1993;329(10):673-82.
16. Gerber Y, Rosen LJ, Goldbourt U, Benyamini Y, Drory Y, Infarction ISGoFAM. Smoking status and long-term survival after first acute myocardial infarction: A population-based cohort study. J Am Coll Cardiol. 2009;54(25):2382-7.
17. Rakowski T, Siudak Z, Dziewierz A, Dubiel JS, Dudek D. Impact of smoking status on outcome in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. J Throm Thrombolysis. 2012;34(3):397-403.
18. Robertson JO, Ebrahimi R, Lansky AJ, Mehran R, Stone GW, Lincoff AM. Impact of cigarette smoking on extent of coronary artery disease and prognosis of patients with non–ST-segment elevation acute coronary syndromes: an analysis from the ACUITY trial (Acute Catheterization and Urgent Intervention Triage Strategy). JACC Cardiovasc Inter. 2014;7(4):372-9.
19. Juliard J-M, Golmard JL, Himbert D, Feldman LJ, Delorme L, Ducrocq G, et al. Comparison of hospital mortality during ST-segment elevation myocardial infarction in the era of reperfusion therapy in women versus men and in older versus younger patients. Am J Cardiol. 2013;111(12):1708-13.
20. Arbel Y, Matetzky S, Gavrielov-Yusim N, Shlezinger M, Keren G, Roth A, et al. Temporal trends in all-cause mortality of smokers versus non-smokers hospitalized with ST-segment elevation myocardial infarction. Int J Cardiol. 2014;176(1):171-6.
21. Arbel Y, FitzGerald G, Yan AT, Tan MK, Fox KA, Gore JM, et al. Temporal trends in all-cause mortality according to smoking status: Insights from the Global Registry of Acute Coronary Events. Int J Cardiol. 2016;218:291-7.
22. Aune E, Røislien J, Mathisen M, Thelle DS, Otterstad JE. The" smoker's paradox" in patients with acute coronary syndrome: a systematic review. BMC Med. 2011;9(1):97.
23. Critchley JA, Capewell S. Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. JAMA. 2003;290(1):86-97.
24. Volpp KG, Troxel AB, Pauly MV, Glick HA, Puig A, Asch DA, et al. A randomized, controlled trial of financial incentives for smoking cessation. N Engl J Med. 2009;360(7):699-709.

Cite this article as: Hirapur I, Setty S, Rajendran R, Nanjappa M. Angiographic patency of streptokinase in STEMI patients: smokers vs. non-smokers. Int J Res Med Sci 2018;6:3813-8.