Indian Heart Journal xxx (2016) xxx–xxx

Contents lists available at ScienceDirect

Indian Heart Journal

journal homepage: www.elsevier.com/locate/ihj

Original Article

Predictors of no- reflow during primary angioplasty for acute myocardial infarction, from Medical College Hospital, Trivandrum

Sabin Padmajan, Alummoottil George Koshy, Prabha Nini Gupta*, Sanjai Pattu Valappil, Sivaprasad Kunjukrishanpilla, Praveen Velappan, Vellikat Velayudhan Radhakrishnan

Medical College Hospital, Trivandrum, 695011, India

ARTICLE INFO

Article history:
Received 1 September 2016
Accepted 12 December 2016
Available online xxx

Keywords:
No-reflow
Primary angioplasty
Predictors

ABSTRACT

Background: Primary angioplasty (PCI) for acute myocardial infarction is associated with no-reflow phenomenon, in about 5–25% of cases. Here we analysed the factors predicting no reflow .
Methods: This was a case control study of consecutive patients with acute myocardial infarction who underwent Primary PCI from August 2014 to February 2015.
Results: Of 181 patients who underwent primary PCI, 47 (25.9%) showed an angiographic no-reflow phenomenon. The mean age was 59.19 ± 10.25 years and females were 11%.
Univariate predictors of no reflow were age >60 years (OR = 6.146, 95%CI 2.937–12.86, P = 0<0.001), reperfusion time >6 h (OR = 21.94, 95%CI 9.402–51.2, P = < 0.001), low initial TIMI flow (≤1) (OR = 12.12, 95%CI 4.117–35.65, P < 0.001), low initial TMPG flow (≤1) (OR = 36.19, 95%CI 4.847–270.2, P < 0.001) a high thrombus burden (OR = 11.04,95%CI 5.124–23.8, P < 0.001), a long target lesion (OR = 8.54, 95%CI 3.794–19.23, P < 0.001), Killip Class III/IV(OR = 2.937,95%CI 1.112–7.756,P = 0.025) and overlap stenting (OR = 3.733,95%CI 1.186–11.75,P = 0.017).
Multiple stepwise logistic regression analysis predictors were: longer reperfusion time > 6 h (OR = 13.844, 95%CI 3.214–59.636, P = <0.001), age >60 years (OR = 8.886, 95%CI 2.145–36.80, P = 0.003), a long target lesion (OR = 8.637, 95%CI 1.975–37.768, P = 0.004), low initial TIMI flow (≤1) (OR = 20.861, 95%CI 1.739–250.290, P = 0.017).
Conclusions: It is important to minimize trauma to the vessel, avoid repetitive balloon dilatations use direct stenting and use the shortest stent if possible.

1. Introduction

Primary angioplasty is an effective treatment for myocardial infarction in that it effectively and rapidly opens up the infarct related artery and provides sufficient information about the disease in the other major epicardial coronary arteries. In spite of its effectiveness in certain patients and in spite of having a TIMI 3 flow, patients experience a phenomenon called no-reflow. This phenomenon is associated with arrhythmias, poor in-hospital survival and poor one year survival[1,2] and has been found to occur in 5 to 25 percent of cases.[3,4]

2. What is no-reflow?

The phenomenon of no-reflow is defined as inadequate myocardial perfusion through a given segment of coronary circulation without angiographic evidence of mechanical vessel obstruction.[7] Occlusion and reperfusion leads to no-reflow.

3. No-reflow in 2016

No-reflow has attracted a great deal of interest, even in 2016. Researchers from London have completed a *meta*-analysis on the use of intravenous and intracoronary adenosine in patients with no-reflow.[5] They calculated the pooled relative risk via a fixed effect *meta*-analysis. They studied the effect of adenosine administration on all-cause mortality, non-fatal myocardial infarction, and congestive heart failure. They analysed 13 randomized controlled trials. In patients who received intra-coronary adenosine, the incidence of no-reflow was reduced and

☆Presented in the National Cardiological Society Conference as an e –poster in Feb 2016 and was awarded the best paper award

* Corresponding author at: 5/2091, Near the Srikrishna Temple, Cheruvekkal, Srikaryam,Trivandrum, 695017, India.
E-mail address: ninigupta@gmail.com (P.N. Gupta).

http://dx.doi.org/10.1016/j.ihj.2016.12.012

the incidence of new onset heart failure was reduced significantly. Intravenous adenosine did not improve the incidence of no-reflow or new heart failure.

Another recent study examined the predictors of no-reflow from a large cohort.[6] The authors analysed data from 781 consecutive patients who had undergone primary angioplasty from 2008 to 2012. Of these, 189 patients had no-reflow. The patients who had no-reflow were older, lower TIMI flows and a higher thrombus score (more than 4). According to the multivariate analysis, the presence of cardiogenic shock, age of more than 60 years, thrombus score of more than 4 and balloon time of more than 360 min were independent predictors of no-reflow.

4. Stenting and no-reflow

17% of patients developed no-reflow immediately after stenting.[6]

4.1. Death and reinfarction

Patients with no-reflow had a higher incidence of death at 12 months. (13% versus 6% $p < 0.003$).[6]

With this background we decided to publish our study on no-reflow.

5. When does no-reflow develop?

Temporary occlusion of the artery, a prerequisite condition for no-reflow, may be produced in the experimental setting occur during reperfusion of an infarct-related artery or following percutaneous coronary intervention.[7,8] No-reflow is associated with abnormal tissue perfusion, and persistent no-reflow is associated with higher clinical complication rates[8,9]. The concept of coronary no-reflow was first described in experimental models in 1966[10] and then in the clinical setting of reperfusion after myocardial infarction in 1985.[10,11]

No-reflow has been documented in 30% of patients after thrombolysis or mechanical intervention for acute myocardial infarction[8,9,12]. Compared to similar patients with adequate reflow, those with no-reflow are more likely to exhibit congestive heart failure early after myocardial infarction and demonstrate progressive left ventricular cavity dilatation in the convalescent stage of the infarction.[8,9] Persistent no-reflow has been associated with increased mortality and a high incidence of recurrent myocardial infarction.[13,14] Hence, the predictors of no-reflow would be helpful in identifying patients at high risk and those with a higher chance of death.

6. Materials and methods

6.1. Aim

To identify the predictors of no-reflow/slow-flow during primary percutaneous coronary intervention in patients with acute myocardial infarction in our institution.

This is a case control study of consecutive patients with acute myocardial infarction who were admitted to MCH Trivandrum and underwent primary PCI from August 2014 to February 2015.

6.2. Inclusion criteria

Patients admitted to MCH Trivandrum with a diagnosis of acute ST elevation myocardial infarction within 12 h of onset of symptoms who underwent primary PCI were included. The patients were classified as those with no-reflow and those without no-reflow.

- Cases: The patients were considered to exhibit a no-reflow phenomenon if blood flow in the IRA (infarct related artery) was a TIMI $\leq$2 flow despite successful dilatation and in the absence of mechanical complications, such as dissection, spasm or extensive angiographically evident distal embolization, at the completion of the procedure.
- Controls: Patients who did not have no-reflow/slow-flow phenomenon and had a TIMI III flow at the completion of the procedure.

Study Site: Medical College Hospital Thiruvananthapuram.

This hospital is a tertiary care government hospital and is an important referral hospital in Kerala; it caters to patients mainly from South Kerala. We care for a large population and see patients from all over South Kerala. Hence, a sample population that is taken from this hospital might be representative of the population in South Kerala.

In all of the patients a detailed history was obtained, and a physical, electrocardiographic, echocardiographic and laboratory examination was performed and the relevant catheterization data were collected prospectively from the Trivandrum MCH cath registry (a computerized registry started in December 2013).

7. Definitions

New myocardial infarction was defined as new ischemic symptoms that lasted >20 min and new or recurrent ST-segment elevation or depression >1 mm in at least 2 contiguous leads that was associated with a > 20% increase in the cardiac biomarker values that was not attributable to the evolution of the index myocardial infarction.

Post-procedural bleeding was considered to be any overt and actionable haemorrhage not related to coronary artery bypass graft with a $\geq$3 g/dl decrease in haemoglobin that required a prompt evaluation by a health care professional and led to an increased level of care. Bleeding was further categorized as access site related or non–access site related according to its relationship to the arterial vascular access.

No-reflow: Angiographic evidence of the reopening of an occluded coronary artery with an acute reduction in coronary flow (TIMI grade 0–1) in the absence of dissection, thrombus, spasm, or high-grade residual stenosis at the original target lesion.

Slow flow: Lesser degrees of flow impairment (TIMI grade 2) are generally referred to as “slow-flow.”

High thrombus burden: was defined as thrombus grade 4 and grade 5.

Long target lesions: were defined as target lesions that were more than 20 mm in length.

8. Laboratory and echocardiographic evaluation

All of the subjects underwent routine investigations that included a haemogram, electrocardiogram, renal function tests and liver function tests at the time of admission to the ICCU. All patients underwent an echocardiogram once they were stabilized.

9. Inclusion criteria

Patients who were at least 18 years of age who presented within 12 h of the onset of chest pain with a STEMI defined as an ST-segment elevation of 1 mm of more in two or more contiguous leads, a new left bundle-branch block, or a true posterior MI with ST-segment depression of at least 1 mm were included in the study

Please cite this article in press as: P. Sabin, et al., Predictors of no- reflow during primary angioplasty for acute myocardial infarction, from Medical College Hospital, Trivandrum, Indian Heart J (2017), http://dx.doi.org/10.1016/j.ihj.2016.12.012