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Original Article

Outcome of primary PCI – An Indian tertiary care center experience

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ABSTRACT

Objective: To assess the feasibility and outcomes of primary percutaneous coronary intervention (PPCI) for ST-segment elevation myocardial infarction (STEMI) in Indian Scenario.
Methods: Between January 2005 and December 2012, consecutive STEMI patients who underwent PPCI within 12 h of onset of chest pain were prospectively enrolled in a PPCI registry. Patient demographics, risk factors, procedural characteristics, time variables and in-hospital and 30 day major adverse cardiovascular events (MACE) [death, reinfarction, bleeding, urgent coronary artery bypass surgery (CABG) and stroke] were assessed.
Results: A total of 672 patients underwent PPCI during this period. The mean age was 52 ± 13.4 years and 583 (86.7%) were males, 275 (40.9%) were hypertensives and 336 (50%) were diabetics. Thirty one (4.6%) patients had cardiogenic shock (CS). Anterior myocardial infarction was diagnosed in 398 (59.2%) patients. The median chest pain onset to hospital arrival time, door-to-balloon time and total ischemic times were 200 (10–720), 65 (20–300), and 275 (55–785) minutes respectively. In-hospital adverse events occurred in 54 (8.0%) patients [death 28 (4.2%), reinfarction 8 (1.2%), major bleeding 9 (1.3%), urgent CABG 4 (0.6%) and stroke 1 (0.14%)]. Nineteen patients with CS died (mortality rate – (61.3%)). At the end of 30 days, 64 (9.5%) patients had MACE [death 35 (5.2%), reinfarction 10 (2.1%), major bleeding 10 (1.5%), urgent CABG 4 (0.6%) and stroke 1 (0.1%)].

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Conclusion: Our study has shown that PPCI is feasible with good outcomes in Indian scenario. Even though the recommended door-to-balloon time can be achieved, the total ischemic time remained long. CS in the setting of STEMI was associated with poor outcomes.

1. Introduction

Coronary artery disease (CAD) is the most common non-infectious disease in India and by 2015, it is estimated to affect over 65 million of its population. Acute ST-segment elevation myocardial infarction (STEMI) is the most dramatic manifestation of CAD with high morbidity and mortality and timely reperfusion therapy has undoubtedly proved to reduce these adverse events.[1] Primary percutaneous coronary intervention (PPCI) is the most effective therapy for STEMI and achieves rapid and more consistent reperfusion with low complication rate when compared to thrombolysis.[2] This prospective, observational study evaluates the feasibility and outcome of PPCI in Indian scenario.

2. Materials and methods

Between January 2005 and December 2012, consecutive STEMI patients who underwent percutaneous coronary intervention (PCI) within 12 h of onset of chest pain were prospectively enrolled in a PPCI registry. This includes all patients admitted directly to coronary care unit (CCU) and those transferred from nearby referral hospitals. Patients with cardiogenic shock (CS) were also included in the registry. Patients who underwent rescue PCI were excluded. The protocol was cleared by institutional ethics committee.

Once a patient was received in the CCU and the diagnosis of STEMI was confirmed, the in-house catheterization laboratory team was notified. All patients underwent brief history taking to rule out any contraindication to dual antiplatelet treatment and a focused clinical examination to assess the need for mechanical ventilation or circulatory support. A screening echocardiography was done to exclude any mechanical complications. After obtaining informed consent, all patients were loaded with 325 mg of aspirin, 300–600 mg of clopidogrel and 40–80 mg of atorvastatin and transferred to the catheterization laboratory as early as possible.

Procedure was performed either through radial or femoral route. Elective intra-aortic balloon pump was inserted in patients with CS. Non-infarct related artery was imaged first with a diagnostic catheter to rule out any critical lesions with compromised blood flow. Then, 70–100 U/kg heparin was administered intra arterially through the sheath to maintain the activated clotting time (ACT) between 250 and 300 s. Infarct related artery (IRA) was engaged with an appropriate sized guiding catheter and the culprit lesion was crossed with non-hydrophilic soft 0.014″ guide wire. After lesion crossing, the TIMI flow and thrombus burden were assessed. If TIMI flow was grade III and thrombus burden was low (TIMI grade 1 or 2), the lesion was stented directly. Conversely, when there was large thrombus burden, aspiration thrombectomy was done using Export aspiration catheter (Medtronic, Minneapolis, Minnesota) and balloon dilatation was done if the lesion was too tight to allow the passage of the stent or when it was difficult to assess the size of the distal vessel. Intracoronary (IC) nitroglycerine was administered when the hemodynamics permitted to exclude any epicardial coronary spasm. IC anti no-reflow medications and GP IIb-IIIa inhibitors were given according to the need. As per the hospital protocol, bare metal stents (BMS) were used in most of the patients and drug-eluting stents (DES) were used when the patient or lesion characteristics were at high risk for restenosis. In case of multi-vessel disease, PCI is limited to IRA unless patient had significant stenosis with less than TIMI III flow in a non-IRA or patient was in cardiogenic shock. Time from pain onset to hospital arrival, door-to-balloon time and total ischemic time were recorded.

Patients were transferred back to CCU post-procedure and arterial sheath was removed when ACT was less than 150 s. Hemodynamically stable patients were transferred to the wards after 24 h and discharged on the third day. At the time of discharge, all the patients were continued on dual antiplatelets, statin, beta-blocker and ACE inhibitor if not contraindicated. In hospital adverse events (death, reinfarction, urgent CABG, bleeding and stroke) were noted and they were followed up for 30 days. At 30 days all patients were reviewed clinically in the out patient department and medications were optimized. Those who did not come to the OPD were contacted through telephone. Patient with multi-vessel disease underwent follow up angiogram between 6 and 8 weeks and had either angioplasty or surgery according to the coronary anatomy.

3. Definitions

STEMI: Angina or anginal equivalent lasting for >20 min and ST-segment elevation of ≥1 mm in ≥2 contiguous leads, or new left bundle branch block, or true posterior MI with ST depression of ≥1 mm in ≥2 contiguous anterior leads.

Cardiogenic shock: Sustained hypotension with systolic blood pressure less than 90 mmHg for at least 30 min, unresponsive to fluid administration and associated with features of tissue hypoperfusion.

Diabetes mellitus: Fasting glucose > 126 mg/dL or on treatment.

Systemic hypertension: Systolic blood pressure >140 mmHg and or Diastolic pressure >90 mmHg, or on treatment.

Dyslipidemia: Fasting cholesterol >200 mg/dL or on treatment.

Multivessel disease: Presence of >50% stenosis in ≥2 epicardial vessels.

Door-to-balloon time: Time from hospital admission to establishment of IRA flow.

Total ischemic time: Time from pain onset to establishment of IRA flow.

Coronary flow: Coronary flow of the infarct related artery was assessed visually by the operator and classified according to the TIMI grading system on a scale of 0–3 both before and after the PCI.

Procedural success: PCI success was defined as achievement of vessel patency to a residual stenosis <30%.

Re-infarction: Recurrent chest pain or ischemic equivalent symptoms lasting ≥30 min and new ECG changes consistent with reinfarction and the next CK-MB (or CK) level measured approximately 8–12 h after the event was at least 50% above the previous level or >3× upper limit of normal, whichever was greater.

Major bleeding: Occurrence of any of the following: intracranial bleeding, intraocular bleeding, retroperitoneal bleeding, access site hemorrhage requiring surgery or a radiological or interventional procedure, hematoma ≥5 cm in diameter at the puncture site, reduction in hemoglobin concentration of ≥4 g/dL without an overt source of bleeding, reduction in hemoglobin concentration of ≥3 g/dL with an overt source of bleeding, re-operation for bleeding, or use of any blood product transfusion.

4. Statistical analysis

All the variables were entered into the Statistical Package for Social Sciences software, version 15 (SPSS Inc) for data analysis. Continuous variables like age, LVEF were presented as means and standard deviations and timing variables like symptom onset to presentation at hospital, door-to-balloon time and total ischemic time were expressed as median and range. Categorical variables like gender, risk factors, cardiogenic shock, left ventricular failure, multi-vessel disease, procedural success, mortality and 30-day outcomes were all reported as percentages.

5. Results

Between January 2005 and December 2012, a total of 5691 PCIs were done at our center, of which 672 (11.8%) were PPCIs. Table 1 shows the demographic and clinical characteristics of the cohort. Males were 86.7%, majority had anterior wall MI (59.2%) and almost half of the patients were diabetics (50%).

Table 2 depicts the angiographic and procedural characteristics. Left anterior descending coronary artery was the most common culprit vessel accounting for 378 patients (56.2%), followed by right coronary artery 209 (30.1%) patients. Multi-vessel disease was present in 252 (37.5%) patients. Stents were deployed in 641 (95.4%) patients and multivessel PCI was performed in 10 (2.1%) patients. Procedure was successful in 639 (95.1%) patients.

Table 3 summarizes timing intervals to PCI. The median symptom onset to hospital arrival time was 200 (10–720) min. The median door-to-balloon time was 65 (20–300) min and less than 90 min in 506 (75.3%) patients. The median total ischemic time was 275 (55–785) min.

16 (2.5%) patients were lost to follow-up. Table 4 summarizes in-hospital and 30-day outcomes and Table 5

Table 1 – Baseline demographic and clinical characteristics.

Characteristic	Number (%)
Age (mean ± SD) (years)	52 ± 13.4
Male gender	583 (86.6)
Past medical history	
Systemic Hypertension	275 (40.9)
Diabetes	336 (50)
Dyslipidemia	103 (15.3)
Current Smoker	169 (25.1)
Family History of CAD	47 (6.9)
Prior MI	34 (5.1)
Prior PCI	14 (2.1)
Prior CVA	4 (0.6)
Prior Renal Dysfunction	13 (1.9)
Clinical characteristics	
Anterior myocardial infarction	398 (59.2)
Cardiogenic Shock	31 (4.6)
Heart rate (mean ± SD) (beats/min)	78 ± 18
Blood pressure (mmHg)	
Systolic (mean ± SD)	127 ± 30
Diastolic (mean ± SD)	82 ± 20
Serum Creatinine (mean ± SD) (mg/dl)	0.95 ± 0.3
CK total (mean ± SD) (IU/L)	2054 ± 2038
Left ventricular ejection fraction (mean) (%)	41.7 ± 20.8%
Intra aortic balloon pump	40 (6)
Mechanical ventilation	26 (5.4)
Temporary pacemaker	52 (7.7)
Hospital stay (mean ± SD) (days)	3.4 ± 1.4

Table 2 – Procedural characteristics.

Characteristic	Number (%)
Culprit Vessel:	
Left anterior descending	378 (56.2)
Left circumflex	70 (10.4)
Right coronary artery	209 (30.1)
LMCA	10 (1.5)
Multi-vessel CAD	252 (37.5)
Pre-PCI TIMI flow	
TIMI 0	409 (60.9)
TIMI I	168 (25)
TIMI II	59 (8.8)
TIMI III	36 (5.4)
Direct stenting	128 (19.0)
PTCA + stenting	513 (76.3)
Plain old balloon angioplasty (POBA)	31 (4.6)
Multi-vessel PCI	10 (2.1)
Total no. of stents used	712
Bare metal stent	488 (68.5)
Drug eluting stent	224 (31.5)
Thrombosuction	228 (33.9)
GP IIb IIIa inhibitor	389 (57.8)
Post-PCI TIMI flow	
TIMI 0	1 (0.1)
TIMI I	32 (4.8)
TIMI II	65 (9.7)
TIMI III	574 (85.4)
Procedural success	639 (95.1)

Table 3 – Timing variables.

Time variable	Number (%)
Symptom onset to hospital arrival time [median (range)]	200 (10–720)
≤120	167 (24.9)
121–240	246 (36.6)
241–360	131 (19.5)
>360	128 (19.0)
Door to balloon time [median (range)]	65 (20–300)
≤60	309 (46)
60–90	197 (29.3)
91–120	94 (14)
>120	72 (10.8)
Total ischemic time [median (range)]	275 (55–785)
<120	62 (9.2)
121–240	219 (32.6)
241–360	203 (30.2)
>360	188 (28)

medications at follow-up. Major adverse events occurred in 64 (9.5%) patients. The 30-day mortality was 5.2% with 61.3% mortality in CS. four (0.6%) patients had emergency coronary artery bypass surgery whereas 4 (0.6%) had emergency surgery for mechanical complications. Major TIMI bleeding requiring transfusion occurred in 10 (1.5%) patients.

6. Discussion

The main observations of our study are: (1) overwhelming majority of patients were males (86.3%), (2) diabetes was the most common risk factor (50%), (3) the median door-to-balloon time was within international recommendations (60 min), (4) the median total ischemic time was 275 min, (5) the overall 30-day mortality was 5.2% and 61.3% in the CS subset.

Prompt restoration of blood flow in the occluded coronary artery and rapid establishment of myocardial perfusion form the basis of STEMI therapy. Even though both thrombolysis and PPCI have been proven to achieve these goals effectively, PPCI has outperformed thrombolysis in many respects. First, thrombolysis restores the IRA patency in fewer (40–60%) patients in contrast to PPCI (more than 90%). Secondly, thrombolysis is less effective when total ischemic time exceeds 6 h when thrombus maturation occurs. Thirdly, up to 25% of patients have contraindication to thrombolysis.[3] Finally, improved hard outcomes in terms of death, myocardial infarction, stroke and bleeding with PPCI makes this the preferred therapy in the setting of STEMI.[2] However, the proportion of patients receiving this treatment remains low in India.[4] In the prospective CREATE registry, which enrolled patients with acute coronary syndrome in 80 centers in various parts of the country, PCI was performed in only 8% of the patients presenting with acute STEMI.[5] Even though the number of centers performing PCI increased since then, there is no substantial improvement in the proportion of patients undergoing PPCI.[6]

Our hospital started 24 × 7 PPCI program in 2005 with an in house cardiologist and catheterization laboratory team so that PCI could be started without delay. Management also

Table 4 – 30-day major adverse cardiovascular events (MACE).

Adverse event	Number (%)		
	In-hospital	Follow-up	Total
Death	28 (4.2)	7 (1.0)	35 (5.2)
Death (cardiogenic shock)	19 (61.3)	0 (0)	19 (61.3)
Death (No cardiogenic shock)	9 (1.4)	7 (1.1)	16 (2.5)
Re-infarction	8 (1.2)	2 (0.4)	10 (1.6)
Mechanical complications	4 (0.6)	0 (0)	4 (0.6)
CABG	4 (0.6)	0 (0)	4 (0.6)
Major bleeding	9 (1.3)	1 (0.1)	10 (1.4)
Stroke	1 (0.1)	0	1 (0.1)
Total event rate	54 (8.0)	10 (1.5)	64 (9.5)

introduced a financial policy in which the patient would only pay a token amount as an initial payment (approximately 10% of the procedure cost) before the PPCI is undertaken and the remaining money can be paid at discharge. Our PCI rates have improved from 18 cases in 2005 to 191 in 2012. Links were created with nearby non-PCI hospitals so that eligible patients after loading with antiplatelets could be transferred without delay. During transfer, the coronary care unit and catheterization laboratory are informed prior and after ruling out mechanical complications by echocardiography patients are transferred to the catheterization laboratory.

The mean age of our cohort was 52 years, considerably younger than patients presenting with STEMI from the west and similar to STEMI subset of CREATE registry.

86% of our patients were males, slightly more than the CREATE subset. In contrast to CREATE, diabetes was more prevalent while smoking and previous history of myocardial infarction was less prevalent in our patients.[5]

Time to treatment or total ischemic time is the most important determinant of not only myocardial salvage but also mortality.[7,8] It starts from the onset of symptoms of myocardial infarction to the initiation of reperfusion. It encompasses two time periods: (1) the time from onset of chest pain to patient arrival at the hospital and (2) the time from patient arrival to the initiation of reperfusion therapy, commonly known as door-to-balloon time.[9,10] The importance of time to treatment has been shown elegantly in animal studies by Reimer et al.[11] Following ligation of left circumflex coronary artery about 45% of the area under risk was necrosed within 40 min; it increased to 67% at 3 h and by 6 h only 16% of the myocardium remained viable. This was later confirmed in human STEMI by using magnetic resonance imaging.[12–14] Thus, the amount of salvageable myocardium is time dependant and gave birth to the concept of "time is muscle". The main determinant of total ischemic time is time

Table 5 – Medications at 30 days.

Medication	Number (%)
Aspirin	616 (99.2)
Clopidogrel	615 (99.0)
Statin	585 (94.2)
Beta-blocker	515 (82.9)
ACE/ARB	532 (85.7)

from pain onset to hospital arrival. In the CREATE registry the median pain onset to hospital arrival time was 360 min, nearly twice the current reported time in developed countries. The various reasons noted for the delay were lack of awareness, paucity of transport facilities, financial difficulties, and inaccurate diagnosis.[5] With such total ischemic times, when they undergo reperfusion, most of the myocardium would be infarcted. Moreover, thrombolysis, the most common reperfusion strategy in India, is largely in effective. Even though, most of our patients come from nearby areas, the median pain onset to hospital arrival was 200 min.

Door-to-balloon time, the second part of time to treatment is the standard metric used to assess hospitals capability to manage STEMI with mechanical reperfusion. Both ACC and ESC propose a door-to-balloon time of 90 min or PCI related delay of 60 min as standard as beyond which the benefit of PPCI over fibrinolysis is lost.[15,16] We could achieve this in nearly 75.3% of the patients. 46% of the patients had door-to-balloon time of less than 60 min. Though we had an in-house catheterization team with an aim to achieve the optimal door-to-balloon time in all patients, for the most part time was lost in patient making the decision about the revascularization strategy.[17]

The procedural success in our study was 95.1%. The primary outcomes (death, reinfarction, urgent CABG, major bleeding and stroke) occurred in 64 (9.5%) patients. The overall 30-day survival was 94.8%. 16 patients out of 641patients without cardiogenic shock died at 30 days with the mortality rate of 2.5%, which is similar to outcomes in studies of primary angioplasty from both developing countries and the west.[18,19] 31 patients presented to us with CS, of which 19 died with a mortality rate of 61.3%, which was slightly more than the mortality in SHOCK study.[20] Most of our patients 589 (87.7%) were discharged within 3days. Follow-up data is available in 97.4% of the patients. Almost all the patients were on dual anti-platelets and a high number of patients were on statin, beta-blocker and ACE-inhibitor or ARB.

7. Limitations

This is a single center observational study conducted in a tertiary care hospital with limited applicability to the rest of the country. Patients were followed up for only 30 days.

8. Conclusion

Our study has shown that PPCI is feasible with good outcomes in Indian scenario. Even though the recommended door-to-balloon time can be achieved in most of the patients the total ischemic time remained long. Increased public awareness needed to improve outcomes in acute MI.

Conflicts of interest

All authors have none to declare.

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Book Review

Manual of 3D Echocardiography Amuthan V. ed. Jaypee brothers medical publishers [P] ltd, New Delhi 2013.

Three-dimensional (3D) echocardiography has rapidly evolved into a useful imaging modality since its advent on the clinical horizon from the research labs about a decade ago. From primarily being considered as a modality which only offered an improved image quality and did not have any major quantitative possibilities, real time 3D echocardiography has come a long way in a relatively short time span. It has been shown to be reliable, reproducible and accurate. Volumetric analysis has been simplified and shown to be comparable to the current gold standard – cardiac MRI. The objectivity introduced, ease of use, coupled with excellent reproducibility has come as a boon for measuring volumes and LV ejection fraction – one of the most useful prognostic parameter in cardiology. In valvular heart disease, 3D derived planimetry of the mitral valve is the current echocardiographic gold standard in mitral stenosis. The repair revolution in mitral valve regurgitation is driving on due to the views provided by 3D echocardiography. In the cath lab too, the percutaneous interventions like transcatheter aortic valve implantation, Mitra-clip, atrial septal defect closure, paravalvular regurgitation closure are all being facilitated by the unique perspectives provided by real time 3D transesophageal echocardiography.

Unfortunately, in spite of the obvious advantages offered by the technique, its use has not spread in the country to the extent possible. The physicians, accustomed over the years to viewing the two-dimensional (2D) images, seem intimidated by the new technique and its nuances. This is partly because of lack of good quality texts dealing with the subject and its application in day to day clinical practice. There has thus been a felt need among the echocardiographers to understand this new modality. This is evident in the interest generated and the attendance in the various workshops conducted on 3D echocardiography.

This book, "Manual of 3D Echocardiography" put together by Dr V Amuthan and his esteemed colleagues, thus could not have been more aptly timed. An eminent cardiologist of the country, Dr Amuthan is an emeritus professor at the Tamil Nadu MGR Medical University, Chennai. He was formerly the head of the department at Madurai Medical College in Tamil Nadu. He is also the Vice President of the Indian academy of echocardiography. Even at this age, he has tremendous interest in the field of echocardiography, particularly, 3D echocardiography, and is a regular at various conferences and events in cardiology. It came as no surprise therefore, that he thought of taking up this task of writing a manual of 3D echocardiography.

The book begins with chapters on the scope of 3D echocardiography and its various modes. There is a description of equipment and the unique features of 3D imaging like slicing and cropping. The book then moves on to explain the role of 3D echo in various clinical conditions with chapters on rheumatic heart disease, coronary artery disease and congenital heart disease with abundant illustrations and references on 3D transthoracic and transesophageal echocardiography. There are separate chapters on 3D TEE guidance in interventions and on device closure of atrial septal defects, a field where 3D TEE is fast becoming indispensable. At the end of the book, there is an atlas of 3D echo images covering some of the common cardiac conditions such as dissection flaps, masses, aortic root abscesses etc. The book is accompanied by 3 DVDs containing video recordings of the lectures on 3D echocardiography and workshops demonstrating the use of 3D echocardiography in cardiac interventions such as balloon mitral valvotomy.

However, there were just a few points which we found worth mentioning. The images in the atlas are not labelled, which makes it somewhat difficult for a beginner to identify the lesions. In addition, a mention of the mode of 3D echo used to obtain these images would have been welcome. In the DVDs, inclusion of 3D clips of various disease states would have been very useful. This would have added to the information needed by the interested readers in understanding the 3D images.

All in all, a good beginning for the texts on 3D echocardiography, in our country!

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