PERIPROCEDURAL MYOCARDIAL INFARCTION FOLLOWING PERCUTANEOUS CORONARY INTERVENTION AT CHARLOTTE MAXEKE JOHANNESBURG ACADEMIC HOSPITAL.


Original published work submitted to the Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, in fulfilment of the requirements for the degree of Master of Medicine (Internal Medicine)

18 October, 2017
TABLE OF CONTENTS

DECLARATION ii
DEDICATION iii
PRESENTATIONS iv
ACKNOWLEDGEMENTS v
PUBLISHED MANUSCRIPT
APPENDIX A – RESEARCH PROTOCOL
APPENDIX B – ETHICS CLEARANCE CERTIFICATE
Declaration

Student’s contribution to article and agreement of co-authors

I, Nqoba Israel Ts Abedze, student number 0101869D, declare that this Research Report is my own work and that I contributed adequately towards research findings published in the article stated below which are included in my Research Report.

Signature of Student...........................................Date...........................................

Name of Primary Supervisor..................................................Signature of Primary Supervisor ..................Date...........................................

Agreement by co-authors: By signing this declaration, the co-authors listed below agree to the use of the article by the student as part of his Research Report.

Article Title: Periprocedural myocardial infarction during percutaneous coronary intervention in an academic tertiary centre in Johannesburg

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<table>
<thead>
<tr>
<th>Authors</th>
<th>Name</th>
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<td>2nd</td>
<td>Kier McCutcheon</td>
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<td>12th</td>
<td>Pravin Manga</td>
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</table>

Comments by Primary Supervisor:

Student made a huge, unique and valuable contribution to a world of medical knowledge through the research contribution to the article and my sincere thanks to you for your research contribution on the above-mentioned article

Prof 1

Manga
To my parents, Daniel and Monica Tsabedze.
To my brothers; Sibusiso, Thembinkosi, Benson, Patrick, Thabiso, Sidumo, Mpumelelo and Simanga.

Thank you all for your unwavering support.
You are appreciated.
Presentations

1. Study Poster (Abstract) was presented at the 16th Annual South African Heart Association Congress held on 25 – 28 October 2015 at the Sun City Resorts, Rustenburg, North West Province. The abstract was subsequently published in the Journal of the South African Heart Association.

2. An oral presentation was done at the University of the Witwatersrand, Faculty of Health Sciences Research Day, held on the 1st of September 2016. I presented my MMed research findings on “periprocedural myocardial infarction in Johannesburg”.
Acknowledgements

I give thanks to Jehovah, God Almighty from whom I have received the strength and courage to persevere and see this project to completion.
Thank you to my supervisor Professor Pravin Manga for his guidance, support and believing in my capability to succeed.
I am grateful to the entire team at the Charlotte Maxeke Johannesburg Academic Hospital for their patience with me while collecting research data and assisting me. Finally, I thank my family and friends for standing by me throughout this remarkable journey.
Periprocedural myocardial infarction during percutaneous coronary intervention in an academic tertiary centre in Johannesburg

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ABSTRACT

Background: Percutaneous coronary intervention (PCI) is effective therapy for significant atherosclerotic coronary artery disease. Despite medical and technological advances in PCI, periprocedural myocardial infarction (PMI) remains a common complication. The frequency and factors associated with PMI have been well investigated in the developed world, yet there is a paucity of data from the developing world, especially Sub-Saharan Africa.

Methods: We prospectively enrolled 153 adult patients undergoing PCI at the Charlotte Maxeke Johannesburg Academic Hospital from the 1st of February 2014 to 31st October 2014. Periprocedural Creatinine Kinase-MB and hs-Troponin I were routinely measured before PCI and at 16–24 h post-procedure. The third universal definition of myocardial infarction was used to define a PMI event.

Results: 152 participants met the inclusion criteria and were analysed for PMI. 70.4% participants were male. The mean age was 58.8 (SD 10.9) years old. Sixteen (10.5%) participants fulfilled the criteria for PMI. Side branch pinching with preserved TIMI III flow was noted in 62.5% of PMI cases. Duration of procedure (p = 0.007), right coronary artery intervention (p = 0.042) and total stent length (p = 0.045) were independently associated with PMI.

Conclusion: PMI occurred in 10.5% of cases undergoing PCI. This is consistent with the prevalence of PMI internationally. Larger multicentre studies are required in our demographic region to further define relevant predictors and outcomes associated with PMI.

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1. Introduction

Coronary artery disease (CAD) has the highest global burden of morbidity and mortality [1,2]. This is also true for the developing world where there has been significant urbanisation [3,4]. Percutaneous coronary intervention (PCI) is a widely accepted therapeutic modality for physiologically significant CAD [5]. Periprocedural myocardial infarction (PMI) is a common complication of PCI and well documented in developed countries [6]. However, there is a paucity of data from developing regions, especially in Sub-Saharan Africa on the prevalence of PMI despite an increasing incidence of CAD and concomitant increase in PCI.

The aim of the current study was to define the local incidence of PMI, to identify relevant risk factors in our study population associated with PMI, and to compare findings to those reported in developed regions.

2. Methods

2.1. Study design and population

This observational study was conducted from the 1st of February 2014 to 31st October 2014. One hundred and fifty-three consecutive
and eligible patients undergoing PCI at the Charlotte Maxeke Johannesburg Academic Hospital (CMJAH), Johannesburg, South Africa were prospectively recruited. This hospital is situated in the heart of Johannesburg with a cardiology unit that functions as a referral point for primary and secondary hospitals without cardiac catheterisation capabilities. These referring hospitals are generally in the periphery of Johannesburg and outskirts of the Gauteng province (18.176km²). As such, the majority of acute coronary syndrome patients referred are first treated medically in their local hospitals and then transferred to CMJAH for coronary angiography.

The study eligibility criteria included any consenting adult in whom PCI was planned. A baseline serum concentration of creatinine kinase myocardial band (CK-MB) mass and highly sensitive troponin 1 (hs-TnI) (ADVIA centaur Tnl –Ultra assay, Siemens Healthcare Diagnostics Inc., NY, USA) were collected after diagnostic coronary angiography, prior to PCI. A second cardiac biomarker was acquired at 16–24 h post PCI in the coronary care unit. All cardiac biomarkers were processed in the same local laboratory (National Health Laboratory Services).

The antiplatelet regime in the cardiology unit follows recommendations of the European Society of Cardiology (ESC) guidelines on myocardial revascularisation [5]. Dual antiplatelet therapy (DAPT) is given to patients with stable coronary artery disease (SCAD), non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI). All loading doses were given on admission to the unit for all acute coronary syndromes and given pre-PCI for SCAD requiring PCI.

In general, patients are loaded with aspirin 300 mg per os (p.o.) if not on prior or long-term aspirin therapy. This is continued with 75 mg of aspirin p.o. daily. A loading dose of 600 mg of clopidogrel is given to DAPT naïve patients or 300 mg to patients previously on clopidogrel. This is then continued with 75 mg of clopidogrel p.o. daily. The DAPT is maintained for 6 months for SCAD and for a year for acute coronary syndromes. The cardiology unit does not have access to prasugrel, ticagrelor, bivalirudin and GP IIb/IIIa inhibitors. Unfractionated heparin at a dose of 70–100 U/Kg was the anticoagulation therapy used during PCI.

The third universal definition of myocardial infarction Type 4 was used to define PMI [7]. The primary end-point (peri-procedural myocardial infarction) was considered when the 16–24-h post PCI cardiac biomarker was elevated by more than five times the 99th percentile upper reference limit (URL) for the local laboratory. This cut-off value was used for patients who had a normal cardiac biomarker baseline (pre-PCI troponin <99th URL). A new elevation in the cardiac biomarker, of >20% of the baseline, was used as a “rule-in” criteria for patients who demonstrated a cardiac biomarker level that was elevated above the 99th percentile URL at baseline, but stable or declining on serial measurements prior to the diagnostic coronary angiography [7]. A diagnosis of PMI was then confirmed by the presence of new periprocedural & post-procedural ischaemic ECG changes or with identifiable angiographic findings consistent with a procedural complication [7].

According to the third universal definition of myocardial infarction type 4a, demonstrating early troponin rise is not as critical as the peak troponin level reached (comparable to the baseline troponin level) in order to rule-in a possibility of PMI. The diagnosis of PMI does not solely rely on the initial cardiac biomarker elevation, instead it compares the magnitude of maximal elevation to baseline levels [7]. A cardiac biomarker measurement strategy designed to detect the peak cardiac biomarker level is therefore more appropriate, and likely to yield more positive results of PMI detection. Based on this rationale, we decided to do cardiac biomarker measurements at 16–24 h post PCI. This strategy has also been successfully used by Zemanek et al. [8].

Patient characteristics were assessed by a detailed medical history and physical examination performed during hospitalisation. PCI was performed according to current guideline management recommendations [5]. The number of treated lesions, duration of inflations, inflation pressure and type of stent were determined individually by the operators. Interventional success was defined as final angiographic residual stenosis <20% of the vessel diameter based on quantitative angiographic evaluation and thrombolysis in myocardial infarction (TIMI) III grade coronary flow.

All clinical, laboratory and angiographic data were assessed and quantified by an independent research team including an experienced cardiologist. The study was approved by the local university ethics committee and complies with the Declaration of Helsinki. All study participants gave written informed consent before participation.

2.2. Statistical analysis

All statistical analyses were generated using STATA version 13.1 (StataCorp, Texas). Continuous variables were expressed as mean ± standard deviation (SD) or as medians and interquartile ranges and discrete variables reported as percentages. Odds ratios (OR) are presented along with their 95% confidence interval (CI). A 2-tailed p value of <0.05 was considered statistically significant. Differences between groups were assessed using the unpaired Student t-test for continuous variables. Categorical variables were analysed using the Chi-square test. Both univariate and multivariate logistic regression analyses were performed. The following parameters were included in the multivariate logistic regression analysis: age, duration of procedure, right coronary artery intervention, C-reactive protein and total stent length. These variables were selected according to known important risk factors for PMI. Study data were collected and managed using REDCap (Research Electronic Data Capture) electronic data capture tools hosted at the University of the Witwatersrand, Johannesburg, South Africa [9].

3. Results

During the 10-month study period 153 participants were recruited. One patient was excluded from the analysis due to a laboratory technical error. (Fig. 1).

Using the third universal definition of myocardial infarction related to PCI (Type 4a), the incidence of PMI in this study was found to be 10.5% (n = 16). There were no significant demographic or anthropometric differences between those who developed PMI and those who did not (Table 1).

The mean age of the study population was 58.8 (SD 10.9) years, 70% of whom were men. There were only 29 (19.1%) black participants in the overall study population. The baseline clinical characteristics in those with and without PMI were also comparable with no significant differences (Table 2). The duration of hours from the onset of reported chest pain to the start of coronary angiography were 43.9 (22.5–126.25) and 106.5 (47.7–106.5), median and interquartile ranges for the STEMI and NSTEMI subgroups, respectively. The procedural results are summarised in Table 3.

Multivariate analysis (Table 4) was performed to identify independent predictors of PMI. In this model, none of the variables showed statistical significance for association with PMI. Of the 16 patients with PMI, 10 of these had side branch compromise, 2 had acute stent thrombosis, 2 distal embolisation and 2 had a flow limiting coronary dissection (Table 5). Table 6 summarises the ECG changes that were associated with these complications.

There were only 2 deaths during PCI which were associated with PMI. The first was a 80 year old male with a high risk non-ST segment elevation myocardial infarction. Coronary angiography revealed triple vessel disease with a SYNTAX score [10] of 29. He declined coronary artery bypass grafting and underwent multivessel PCI of his left circumflex (LCx) and left anterior descending (LAD) coronary artery. While PCI of the proximal LAD artery was being performed, he
complicated with hyper-acute stent thrombosis of his left circumflex artery. Despite revascularisation of the circumflex artery, he deteriorated and demised.

The second was a 42 year old female who presented with an inferior ST segment elevation myocardial infarction due to acute stent thrombosis (PCI performed 9 days previously) from poor compliance to antiplatelet therapy. Coronary angiography revealed total occlusion of the proximal right coronary artery (RCA) with high thrombus burden. Revascularisation of her RCA complicated with proximal and distal thrombus embolisation. She arrested and cardiopulmonary resuscitation was unsuccessful.

4. Discussion

The present study is the first systematic study in Sub-Saharan Africa investigating the incidence of PMI according to the third universal

<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall population (n = 152)</th>
<th>Peri-procedural Myocardial Infarction</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>58.8 SD 10.9</td>
<td>58.3 SD 10.7</td>
<td>0.220</td>
</tr>
<tr>
<td>Gender:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>107 (70.4)</td>
<td>96(69.6)</td>
<td>0.879</td>
</tr>
<tr>
<td>Ethnicty</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>White</td>
<td>64 (42.1)</td>
<td>59(43.4)</td>
<td>0.316</td>
</tr>
<tr>
<td>Indian</td>
<td>47 (30.9)</td>
<td>39(28.7)</td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>29 (19.1)</td>
<td>26(19.1)</td>
<td></td>
</tr>
<tr>
<td>Mixed Ancestry</td>
<td>12 (7.9)</td>
<td>12(8.8)</td>
<td></td>
</tr>
<tr>
<td>Height (meters)</td>
<td>1.7 SD 0.1 (n = 142)</td>
<td>1.7 SD 0.1 (n = 126)</td>
<td>0.849</td>
</tr>
<tr>
<td>Weight (kilograms)</td>
<td>77.7 SD 17.8 (n = 147)</td>
<td>77.4 SD 17.3 (n = 131)</td>
<td>0.561</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.0 SD 6.3 (n = 142)</td>
<td>27.9 SD 6.4 (n = 126)</td>
<td>0.665</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.9 SD 0.2 (n = 142)</td>
<td>1.9 SD 0.2 (n = 126)</td>
<td>0.638</td>
</tr>
</tbody>
</table>

Data are shown as mean, standard deviation (SD) for continuous variables and absolute numbers (percentage) for dichotomous variables.

BMI, body mass index; BSA, body surface area.
definition of myocardial infarction [7]. Despite operating in a resource limited centre with restricted access to newer antiplatelet agents and novel coronary imaging modalities which have been shown to prevent and better predict PMI [11–14], the incidence of PMI in our cohort was 10.5%. This is consistent with the prevalence of PMI internationally. Our findings are reassuring in light of the increasing frequency of PCI being performed in our population.

Duration of procedure, total stent length and RCA intervention were the only risk factors associated with the development of PMI. In our study a longer duration of procedure could have been an indirect measure of lesion complexity or a measure of interventional operator competence. However, in our analysis we did not find an association between coronary artery lesion complexity and PMI. This has been demonstrated by Van Gaal et al. [15] who showed that a higher incidence of PMI correlated with a higher Syntax score [10]. Hoole et al. [16] has previously demonstrated the association between coronary artery stent length and PMI.

A recently published systematic review on predictors of PMI based on 11 prospective PCI studies with 23,604 patients, also found that higher lesion and procedural-related risk profiles were associated with PMI [17]. Left anterior descending artery disease, left main disease, bifurcation lesions, lesions (>20 mm) and number of stents deployed were found to be independent predictors of PMI [17]. These risk factors have been associated with a greater incidence of side branch occlusion (type 1 PMI) and structural or functional microvascular obstruction (type 2 PMI) [18–20].

Our study is unique in that we found revascularisation of the RCA to be associated with PMI. Previous studies have not observed this association [17]. PCI of the RCA is generally thought to be less complicated compared to the other coronary arteries. Of the 16 patients with PMI, 9 involved interventions of the RCA. Of these 5 related to side branch compromise; 2 patients had distal embolisation; 1 had a flow limiting dissection and 1 patient had an acute stent thrombosis. In our cohort, the low threshold to stent across side branches in the RCA probably accounts for our findings that RCA interventions are associated with PMI.

In this study side branch compromise was observed in 62.5% of all cases complicating with PMI. This finding is well documented in previous studies and is thought to be related to the length of the carina, carina deformation and plaque shift [21,22]. The determination of fractional flow reserve in a compromised side branch after main branch stenting has previously shown that generally these lesions are not physiologically significant [23]. Despite this finding, side branch compromise remains the most common cause of PMI [17,24].

Although our unit provides a clinical service to a predominantly black population, only a fifth of the study population was black. This correlates with previously published African CAD data suggesting that the prevalence of CAD in black African people is relatively low compared to their white and Indian counterparts [25,26]. However as more black African people become urbanised their risk of developing CAD has increased compared to reported data from non-urbanised black African communities [27].

The human immunodeficiency virus (HIV) infection is highly prevalent in South Africa [28]. It has been shown that HIV positive patients taking anti-retroviral medication have an increased risk of developing CAD [29,30]. In our study only 4 participants were HIV positive and none of them developed PMI. The anticipated increased burden of CAD due to HIV and combination antiretroviral therapy (cART) has thus far not been noticed in our population. This could be related to the very low prevalence of CAD amongst the black population despite the high prevalence of HIV and increasing access to cART.

The study limitations include a relatively small sample size. There is also the possibility, albeit very unlikely, of missing cardiac biomarker elevation in the interval from PCI to biomarker evaluation 16–24 h later.

Table 2
Baseline clinical characteristics of patients according to periprocedural myocardial infarction.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall population (n = 152)</th>
<th>Peri-procedural Myocardial Infarction</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No (n = 136) (89.5%)</td>
<td>Yes (n = 16) (10.5%)</td>
<td></td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>9 (4–45) (n = 141)</td>
<td>9 (4–48) (n = 126)</td>
<td>12 (4–19) (n = 15)</td>
</tr>
<tr>
<td>TIMI Score</td>
<td>3.6 SD 1.7 (n = 122)</td>
<td>3.6 SD 1.7 (n = 113)</td>
<td>3.1 SD 1.2 (n = 9)</td>
</tr>
<tr>
<td>WCC (10^9/L)</td>
<td>10.5 SD 2.7 (n = 149)</td>
<td>10.6 SD 3.8 (n = 133)</td>
<td>9.2 SD 2.5</td>
</tr>
<tr>
<td>Total Cholesterol (mmol/L)</td>
<td>4.2 SD 1.3</td>
<td>4.3 SD 1.3</td>
<td>3.6 SD 1.3</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>0.9 SD 0.3 (n = 151)</td>
<td>0.9 SD 0.3 (n = 135)</td>
<td>0.8 SD 0.3</td>
</tr>
<tr>
<td>LDL-C (mmol/L)</td>
<td>2.6 SD 1.0 (n = 148)</td>
<td>2.7 SD 1.0 (n = 132)</td>
<td>2.2 SD 1.0</td>
</tr>
<tr>
<td>Pre PCI eGFR (ml/min/1.73m^2)</td>
<td>90.5 SD 30.8 (n = 146)</td>
<td>91.1 SD 31.0 (n = 130)</td>
<td>85.7 SD 30.3</td>
</tr>
<tr>
<td>16–24 h Post PCI eGFR (ml/min/1.73m^2)</td>
<td>84.8 SD 29.1 (n = 140)</td>
<td>85.2 SD 30.0 (n = 129)</td>
<td>79.5 SD 16.0 (n = 11)</td>
</tr>
<tr>
<td>Admission LVEF (%)</td>
<td>51.6 SD 12.1 (n = 148)</td>
<td>51.6 SD 11.9 (n = 133)</td>
<td>51.3 SD 14.3 (n = 15)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>38 (25.0)</td>
<td>33 (24.3)</td>
<td>5 (31.3)</td>
</tr>
<tr>
<td>HBA1c</td>
<td>9.4 SD 2.3 (n = 32)</td>
<td>9.7 SD 2.4 (n = 27)</td>
<td>7.7 SD 1.2 (n = 5)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>97 (63.8)</td>
<td>86 (63.2)</td>
<td>11 (68.8)</td>
</tr>
<tr>
<td>HIV Positive</td>
<td>4 (2.6)</td>
<td>4 (2.9)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Current Smoker</td>
<td>99 (65.1)</td>
<td>92 (67.6)</td>
<td>7 (43.8)</td>
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<tr>
<td>NYHA Classification</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>115 (75.7)</td>
<td>102 (75.0)</td>
<td>13 (81.3)</td>
</tr>
<tr>
<td>II</td>
<td>30 (19.7)</td>
<td>28 (20.6)</td>
<td>2 (12.5)</td>
</tr>
<tr>
<td>III</td>
<td>2 (1.3)</td>
<td>2 (1.5)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>IV</td>
<td>5 (3.3)</td>
<td>4 (2.9)</td>
<td>1 (6.3)</td>
</tr>
<tr>
<td>Acute coronary syndrome</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STEMI</td>
<td>119 (78.3)</td>
<td>110 (80.9)</td>
<td>9 (56.2)</td>
</tr>
<tr>
<td>NSTEMI</td>
<td>66 (43.6)</td>
<td>80 (58.8)</td>
<td>6 (37.5)</td>
</tr>
<tr>
<td>Non-acute coronary syndrome</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta blockers</td>
<td>85 (55.9)</td>
<td>73 (53.7)</td>
<td>12 (75.0)</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>88 (57.9)</td>
<td>77 (56.6)</td>
<td>11 (68.8)</td>
</tr>
<tr>
<td>Statins</td>
<td>115 (75.7)</td>
<td>103 (75.7)</td>
<td>12 (75.0)</td>
</tr>
<tr>
<td>Calcium channel blocker</td>
<td>17 (11.2)</td>
<td>14 (10.3)</td>
<td>3 (18.8)</td>
</tr>
</tbody>
</table>

Data are shown as mean, standard deviation (SD) for continuous variables and absolute numbers (percentage) for dichotomous variables.

CRP, C-reactive protein; TIMI, thrombolysis in myocardial infarction; WCC, white cell count; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; HBA1c, glycated haemoglobin; ACE, angiotensin converting enzyme.
The small sample size with a low expected incidence of PMI in PCI, may be associated with a potential statistical type II error.

5. Conclusion

The results of this single centre, prospective observational study has found an incidence of PMI to be 10.5%. Despite being in a resource limited environment this is consistent with the prevalence of PMI internationally. Larger multicentre studies are required in our demographic region to further define relevant predictors and outcomes of PMI.

Disclosures

The authors report no relationships that could be construed as a conflict of interest.

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The authors would like to thank Sr. R Zwapano, Sr. M Modiga, Mrs. R Kgomommu, Sr. P Ewing, Sr. V Paton, the catheterisation laboratory staff and the coronary care unit staff for their administrative and logistical support.

Table 3

<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall population (n = 152)</th>
<th>Peri-procedural myocardial infarction</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-PCI Systolic BP (mmHg)</td>
<td>133.0 SD 29.6</td>
<td>133.8 SD 30.3</td>
<td>126.1 SD 22.9</td>
</tr>
<tr>
<td>Pre-PCI Mean BP (mmHg)</td>
<td>97.1 SD 20.3</td>
<td>97.3 SD 20.3</td>
<td>96.3 SD 17.8</td>
</tr>
<tr>
<td>Framingham 10 Year Risk (%)</td>
<td>13.6 SD 9.2</td>
<td>13.9 SD 9.4</td>
<td>10.7 SD 7.4</td>
</tr>
<tr>
<td>Duration of Procedure (minutes)</td>
<td>87.8 SD 30.8</td>
<td>85.6 SD 28.8</td>
<td>102.2 SD 40.3</td>
</tr>
<tr>
<td>Total Stent Length (mm)</td>
<td>25.0 SD 10.5</td>
<td>24.5 SD 10.0</td>
<td>30.1 SD 13.5</td>
</tr>
<tr>
<td>Contrast Media Used (ml)</td>
<td>260.0 SD 77.0</td>
<td>257.3 SD 78.9</td>
<td>283.1 SD 54.5</td>
</tr>
<tr>
<td>Duration of Hospital Stay (days)</td>
<td>3.8 SD 3.3</td>
<td>3.9 SD 3.4</td>
<td>3.4 SD 2.4</td>
</tr>
<tr>
<td>Coronary artery disease distribution</td>
<td>(n = 152)</td>
<td>(n = 136)</td>
<td>(n = 16)</td>
</tr>
<tr>
<td>Single vessel disease</td>
<td>82(54.0)</td>
<td>75(55.2)</td>
<td>7(43.8)</td>
</tr>
<tr>
<td>Double vessel disease</td>
<td>45(29.6)</td>
<td>39(28.7)</td>
<td>6(37.5)</td>
</tr>
<tr>
<td>Triple vessel disease</td>
<td>25(16.5)</td>
<td>22(16.2)</td>
<td>3(18.8)</td>
</tr>
<tr>
<td>Coronary artery treated</td>
<td>LMCA</td>
<td>1(0.7)</td>
<td>1(0.7)</td>
</tr>
<tr>
<td>LAD</td>
<td>74(48.7)</td>
<td>67(49.3)</td>
<td>7(43.8)</td>
</tr>
<tr>
<td>LCx</td>
<td>29(19.1)</td>
<td>27(19.9)</td>
<td>2(12.5)</td>
</tr>
<tr>
<td>RCA</td>
<td>51(33.6)</td>
<td>42(30.9)</td>
<td>9(56.3)</td>
</tr>
<tr>
<td>Other CAD</td>
<td>10(6.6)</td>
<td>9(6.6)</td>
<td>1(6.3)</td>
</tr>
<tr>
<td>Coronary artery lesion site</td>
<td>Ostial</td>
<td>3(2.0)</td>
<td>3(2.2)</td>
</tr>
<tr>
<td>Proximal</td>
<td>79(52.0)</td>
<td>69(50.7)</td>
<td>10(62.5)</td>
</tr>
<tr>
<td>Mid Vessel</td>
<td>75(49.3)</td>
<td>66(48.5)</td>
<td>9(56.3)</td>
</tr>
<tr>
<td>Distal</td>
<td>24(15.8)</td>
<td>22(16.2)</td>
<td>2(12.5)</td>
</tr>
<tr>
<td>Coronary artery lesion complexity</td>
<td>(n = 152)</td>
<td>(n = 136)</td>
<td>(n = 16)</td>
</tr>
<tr>
<td>A</td>
<td>29(19.1)</td>
<td>28(20.6)</td>
<td>1(6.3)</td>
</tr>
<tr>
<td>B</td>
<td>115(75.7)</td>
<td>100(73.5)</td>
<td>15(93.8)</td>
</tr>
<tr>
<td>C</td>
<td>8(5.3)</td>
<td>8(5.9)</td>
<td>0(0.00)</td>
</tr>
<tr>
<td>Stent choice</td>
<td>Drug Eluting Stent</td>
<td>141(92.8)</td>
<td>126(92.7)</td>
</tr>
<tr>
<td>Bare Metal Stent</td>
<td>1(0.7)</td>
<td>1(0.7)</td>
<td>0(0.00)</td>
</tr>
<tr>
<td>Drug Eluting Balloon</td>
<td>15(9.9)</td>
<td>14(10.3)</td>
<td>1(6.3)</td>
</tr>
<tr>
<td>Other Stent</td>
<td>1(0.7)</td>
<td>1(0.7)</td>
<td>0(0.00)</td>
</tr>
<tr>
<td>Intervention success</td>
<td>TIMI III flow and good angiographic result</td>
<td>143(94.1)</td>
<td>129(94.9)</td>
</tr>
</tbody>
</table>

Data are shown as mean, standard deviation (SD) for continuous variables and absolute numbers (percentage) for dichotomous variables.

Table 4

Multivariable logistic regression analysis independent predictors of periprocedural myocardial infarction.

<table>
<thead>
<tr>
<th>Variables</th>
<th>OR</th>
<th>95% CI</th>
<th>Z</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>1.01</td>
<td>0.95–1.07</td>
<td>0.35</td>
<td>0.728</td>
</tr>
<tr>
<td>Smoking (Yes)</td>
<td>0.62</td>
<td>0.58–1.02</td>
<td>-0.76</td>
<td>0.447</td>
</tr>
<tr>
<td>Duration of procedure (min)</td>
<td>1.02</td>
<td>0.99–1.04</td>
<td>1.89</td>
<td>0.061</td>
</tr>
<tr>
<td>Right coronary artery intervention</td>
<td>2.25</td>
<td>0.69–7.29</td>
<td>1.35</td>
<td>0.178</td>
</tr>
<tr>
<td>C– Reactive protein (mg/L)</td>
<td>0.98</td>
<td>0.95–1.00</td>
<td>-1.39</td>
<td>0.163</td>
</tr>
<tr>
<td>Total stent length (mm)</td>
<td>1.01</td>
<td>0.96–1.07</td>
<td>0.4</td>
<td>0.691</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; OR, odds ratio; min, minutes.

Table 5

Causes of periprocedural myocardial infarction.

<table>
<thead>
<tr>
<th>Complication</th>
<th>No of patients (n = 16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Side branch compromise</td>
<td>10 (62.5)</td>
</tr>
<tr>
<td>Acute stent thrombosis</td>
<td>2 (12.5)</td>
</tr>
<tr>
<td>Distal embolisation</td>
<td>2 (12.5)</td>
</tr>
<tr>
<td>Flow limiting coronary dissection</td>
<td>2 (12.5)</td>
</tr>
</tbody>
</table>

All data presented as number (%).

Table 6

Persistent ischaemic periprocedural ECG changes.

<table>
<thead>
<tr>
<th>Complications (n = 16)</th>
<th>Total no of patients with ECG changes (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Side branch compromise</td>
<td>3 (30)</td>
</tr>
<tr>
<td>Acute stent thrombosis</td>
<td>2 (100)</td>
</tr>
<tr>
<td>Distal embolisation</td>
<td>1 (50)</td>
</tr>
<tr>
<td>Flow limiting coronary dissection</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

All data presented as number (%).
Appendix A.

Charlotte Maxeke Johannesburg Academic Hospital
State Patients Annual Angiography and Percutaneous Coronary Intervention (PCI) Count

<table>
<thead>
<tr>
<th>Year</th>
<th>Total Number of Coronary Angiograms</th>
<th>Total Number of PCIs</th>
</tr>
</thead>
<tbody>
<tr>
<td>2009</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2010</td>
<td>200</td>
<td>200</td>
</tr>
<tr>
<td>2011</td>
<td>400</td>
<td>400</td>
</tr>
<tr>
<td>2012</td>
<td>600</td>
<td>600</td>
</tr>
<tr>
<td>2013</td>
<td>800</td>
<td>800</td>
</tr>
<tr>
<td>2014</td>
<td>1000</td>
<td>1000</td>
</tr>
<tr>
<td>2015</td>
<td>1200</td>
<td>1200</td>
</tr>
</tbody>
</table>

Fig. 2. Charlotte Maxeke Johannesburg Academic Hospital state patients’ annual angiography & percutaneous coronary intervention count.

References
Dear Dr Tsabedze

Master of Medicine: Approval of Title

We have pleasure in advising that your proposal entitled *Periprocedural myocardial infarction following percutaneous coronary intervention at Charlotte Maxeke Johannesburg Academic Hospital* has been approved. Please note that any amendments to this title have to be endorsed by the Faculty's higher degrees committee and formally approved.

Yours sincerely

Mrs Sandra Benn
Faculty Registrar
Faculty of Health Sciences
PERIPROCEDURAL MYOCARDIAL INFARCTION FOLLOWING PERCUTANEOUS CORONARY INTERVENTION AT CHARLOTTE MAXEKE JOHANNESBURG ACADEMIC HOSPITAL.

Student: Dr Nqoba Tsabedze Student No. 0101869D Master of Medicine Degree in Internal Medicine

Supervisor: Prof P. Manga – Head of Cardiology, Charlotte Maxeke Johannesburg Academic Hospital

1. Introduction and Extended Literature Review

1.1 Introduction

The very first coronary artery balloon angioplasty is reported to have been performed by Gruntzig in 1977.\(^1\) Subsequently to this, over the past 40 years, there have been significant advances in coronary angiography and intervention. Coronary artery interventional techniques have evolved and improved significantly. There have been considerable device developments, new generation stents and novel antiplatelet therapy which have all proved to reduce the incidence of the primary periprocedural complications associated with percutaneous coronary intervention (PCI).\(^2,3\) Despite these significant advances, periprocedural myocardial infarction remains a common complication of PCI.\(^4\)

Coronary heart disease is predicted to remain the number one cause of morbidity and mortality worldwide until the year 2030, and beyond.\(^5\) This emerging epidemic is
also exponentially increasing in the developing world, where urbanisation has occurred.\textsuperscript{6} Percutaneous coronary intervention has become a prominent therapeutic procedure for atherosclerotic coronary artery disease.\textsuperscript{7} In sub-Saharan Africa the use of this method is also increasing, yet there is no regional data on the safety and complications associated with this procedure.\textsuperscript{8}

1.2 Definition and diagnosis of periprocedural myocardial infarction (PMI)

The definition of PMI has been revised multiple times since its original description. Over the years, it has been modified by changing the preferred cardiac biomarker used to make a diagnosis of PMI and different peak cardiac biomarker elevations were proposed to rule in PMI.\textsuperscript{9,10} The current definition of PMI is from the third universal definition of myocardial infarction. This definition was published in 2012.\textsuperscript{10} According to this definition; you may diagnose PMI (Type 4a MI) when there is a post-procedural increase of a cardiac biomarker greater than five times the 99th percentile upper reference limit (URL) within 24 to 48 hours after the procedure.\textsuperscript{10} A non-elevated baseline (pre-procedural) cardiac biomarker value is required to use this definition.

The global task force recommends the use of highly sensitive cardiac troponins (hs-cTnI or cTnT) to creatinine kinase myocardial band (CK-MB) for the definition of acute myocardial infarction.\textsuperscript{10-12} Cardiac troponins are reported to have an increased sensitivity and specificity than CK-MB and therefore have a better diagnostic
Use of these overly sensitive tests to detect myocardial injury after PCI can be challenging as small increases from the baseline in biomarker levels after PCI are expected and do not necessarily represent a complication.  

Criteria for a new periprocedural injury have also been proposed based on a new troponin rise after an apparently falling pre-procedural pattern. If the baseline troponin is elevated, yet stable or decreasing, a new increase of > 20 percent can be used to define a periprocedural MI event. The clinical challenge is evident when a patient presents with ST-segment elevation MI (STEMI) and undergoes primary PCI. In some of these patients, cardiac biomarkers are not yet elevated or are still rising, thus making it difficult to differentiate whether the elevation is due to the index STEMI or caused by the PCI procedure. Following PCI, an elevation of either troponin or CK-MB above the upper limits of normal has a worse short and long term prognosis.

Over the years, the reliance on cardiac biomarkers alone to clinch a diagnosis of PMI has been abandoned. The third universal definition requires that patients should have at least one of the following also:

i) Symptoms in keeping with myocardial ischaemia

ii) New ischaemic electrocardiography changes

iii) Coronary angiographic findings suggestive of a procedural complication
iv) demonstrating a new loss of viable myocardium or new regional wall motion abnormality on cardiac imaging.

Depending on the consensus definition used at the time of data collection, the incidence of PMI has varied considerably. The recommended choice of a cardiac biomarker used, peak cut-off value used and frequency of blood sampling for cardiac biomarker analysis, all affected the reported incidence of PMI. A recently published large meta-analysis reported an incidence of seven percent measured by CK-MB.

1.3 Risk factors for Periprocedural Myocardial Infarction

1.3.1 Patient-specific risk factors for periprocedural myocardial infarction

The total atherosclerotic burden carried by the patient is an important risk factor for PMI. Generalised atherosclerosis, diffuse coronary artery disease (CAD) and multivessel CAD are all associated with an increased incidence of PMI. Advancing age, chronic kidney disease and anaemia have all been confirmed to promote PMI. Implicated markers of inflammation include a raised pre-procedural C-reactive protein and white blood cell count.

1.3.2 Coronary artery lesion risk factors for periprocedural myocardial infarction

Coronary artery disease risk factors include the left main artery and proximal left anterior descending artery interventions. Furthermore, the more complex the lesion, such as type C lesions and a higher syntax score, the greater the association with
PMI. This is most likely due to prolonged and challenging catheter manipulation required to treat the lesion percutaneously successfully. Due to their friable calibre, saphenous vein graft interventions are also responsible for causing PMI.\textsuperscript{22}

1.3.3 Coronary artery interventional risk factors for PMI

Procedural factors associated with PMI include multivessel PCI, atherectomy, side branch occlusion, dissection, increased thrombus burden, slow flow/no-reflow phenomenon and distal embolisation.\textsuperscript{20} In line with the third universal definition of myocardial infarction, angiographic complications are the most frequently listed and strongest predictors of PMI.\textsuperscript{4}

1.4 Pathophysiology of periprocedural myocardial infarction

Coronary artery interventions by their very nature cause plaque disruption, redistribution, compression and fragmentation. This vessel injury causes atherosclerotic micro emboli which have the potential to cause distal embolism and occludes the epicardial and myocardial microvascular tree.\textsuperscript{25} PMI has two primary forms:

Type 1 – Proximal-type PMI. This type of PMI is mainly due to side branch occlusion of an epicardial artery and is in proximity to the treated atherosclerotic lesion.\textsuperscript{25}

Type 2 – Distal type PMI. This type of PMI is the most common mechanism of PMI. It is due to structural and functional microvascular obstruction of the distal coronary tree. It occurs distal to the treated atherosclerotic lesion.\textsuperscript{25}
1.5 Treatment and prevention of periprocedural myocardial infarction

There are multiple therapies for the prevention of PMI. These include the use of antiplatelet drugs,\textsuperscript{24,26} statins,\textsuperscript{27,28} ischaemic preconditioning, use of adenosine as well as distal embolic protection devices.\textsuperscript{22}

The treatment of peri-procedural MI depends upon identifying and treating the underlying cause. Periprocedural MI is usually silent and not diagnosed during the procedure, but recognised afterwards if cardiac enzymes are routinely measured.\textsuperscript{10} Supportive measures alone are adequate for 1 to 5-fold increase in cardiac biomarker.\textsuperscript{4} Q-wave infarcts and those with CK-MB or troponin >5 URL need coronary intervention therapy. Treatment should follow guidelines for the management of spontaneous STEMI and non-STEMI.\textsuperscript{25}

1.6 Long-term prognosis of periprocedural myocardial infarction

A correlation exists between PMI and future major adverse cardiac events (MACE).\textsuperscript{13} Cardiovascular mortality is proportionally related to the increase in cardiac biomarker level. Post-procedural cardiac biomarker levels > 5 URL are considered significant.\textsuperscript{29}

The latest criteria for diagnosing PMI is the strictest to date and requires the presence of a confirmed complication to diagnose PMI. This definition is likely to improve the association of PMI and MACE outcomes further.\textsuperscript{30}
On average, the Charlotte Maxeke Johannesburg Academic Hospital cardiology unit performs 3 to 4 PCIs per week. Despite this high hospital and operator volume, our local incidence of periprocedural MI still needs to be determined. The aim of our study is therefore to define our local rate of peri-procedural MI. To describe the relevant risk factors identified in our patients undergoing PCI and to correlate our outcomes with the observed levels of cardiac biomarkers and to compare our findings to other centres abroad.

2. STUDY OBJECTIVES

1. Define the incidence of peri-procedural MI at CMJAH.
2. To identify key risk factors associated with peri-procedural MI.
3. To correlate outcomes with the observed levels of cardiac biomarkers.
4. To compare our incidence rate of peri-procedural MI to other centres.

3. METHODS

3.1 Study Design

This will be a prospective review of clinical data of all patients undergoing PCI over a 6-month period at Charlotte Maxeke; with analytical and descriptive elements.

3.2 Study Population and Samples

This study will be conducted in the division of cardiology at the Charlotte Maxeke Academic Hospital in Johannesburg. Data will be collected from all patients.
undergoing PCI in the catheterisation laboratory over a 6-month period targeting a sample size of 100 patients. The study may continue beyond 6 months until a minimum of 100 patients has been reviewed. The population under study is all adult (above 18 years). The sample size will be dependent on the total number of patients on whom PCI is performed.

3.3 Eligibility Criteria

All patients in whom PCI is done are eligible for this study. A baseline cardiac marker is required before PCI. The trend will then be followed post PCI at 16 to 24 hours. Increases of biomarkers greater than 5 X 99th percentile URL will be used to define PCI – related MI (type 4a).10 This is in line with the Universal definition of Myocardial infarctions which has been endorsed academic societies & regulatory bodies. Patients with an elevated cardiac biomarker >99th percentile URL; myocardial re-infarction is defined by a new rise of >20% in serum biomarkers over the last nadir.10 This meets criteria for differences in analytical values (>3SD difference of the variance of the measure).

3.4 Study Tools

All bed letter records/information for the patients undergoing PCI will be identified. Data collection sheets will be used to obtain information from the bed letter. (See appendix)
3.5 Variables

Risk Factors for Developing Periprocedural Myocardial Infarction (PMI)

Patient Factors:

1. Age, gender, ethnicity

2. Co-morbidities: diabetes, hypertension, chronic kidney disease, HIV, peripheral vascular disease, dyslipidaemia, family history of ischaemic heart disease, previous MI, smoking.

3. Clinical presentation: NYHA, STEMI, NSTEMI, TIMI score, C-reactive protein, White cell count, Total cholesterol, LDL, creatinine, CK-MB, Trop I, Left ventricular ejection fraction and pre-angiogram blood pressure.


Angiographic Lesion Factors:

1. Distribution of CAD: single, double or triple vessel disease.

2. Vessel treated: Left main, LAD, LCx, RCA, other branches.

3. Lesion Site: Ostial, proximal, midvessel, distal.

4. Number of treated vessel/patient

5. Number of treated lesion/patient
6. Multivessel PCI

7. Complex lesions (B2/C)

Procedural Factors:

1. Duration of procedure

2. Major dissection / perforation

3. Significant spam / slow / no reflow

4. High thrombus burden

5. Side-branch closure / compromise

6. Distal embolisation

7. Type of stent used.

8. Total length of stent (mm)

9. Total contrast agent (ml)

10. Successful PCI (%)

Cardiac Biomarkers

Trop I & CK-MB levels: pre-PCI, and at 16 to 24hrs Post PCI.

Duration of hospital stay (days)
4. DATA ANALYSIS:

Data will be presented as mean ± 2SD, median and interquartile range, or as a percentage, as appropriate. Descriptive statistics-frequency tables; pie charts; bar graphs will be used to describe demographic and clinical characteristics. Chi-test and Fisher’s exact will be used for categorical data, student t- test for normally distributed continuous variables and Wilcoxon rank sum test (parametric test) for non-normal continuous data. Continuous variables will be tested for normality using histograms and normal quartile plots. STATA version 11 will be used for the data analysis.

5. ETHICS

This study will be conducted at Charlotte Maxeke Academic Hospital in the Department of Cardiology in fulfilment of the requirements of the Master of Medicine degree research report. Ethics approval will be sought from the University of Witwatersrand Human Research Ethics Committee and relevant hospital authorities. Written informed consent will be sought from all patients before they participate in the study. Unique patient identifiers will be used on the data collection sheet to protect the identity of the study participants. Findings will be published in an academic journal and will be used to improve patient care in the Department of cardiology at Charlotte Maxeke Academic Hospital.
6. TIMING

<table>
<thead>
<tr>
<th>TASKS</th>
<th>PROPOSED COMPLETION DATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protocol assessment</td>
<td>August 2012</td>
</tr>
<tr>
<td>Ethics Application</td>
<td>September 2012 to October 2012</td>
</tr>
<tr>
<td>Data Collection</td>
<td>November 2012 to May 2014</td>
</tr>
<tr>
<td>Data analysis</td>
<td>June to July 2014</td>
</tr>
<tr>
<td>Write up - thesis</td>
<td>July to September 2014</td>
</tr>
<tr>
<td>Write up - paper</td>
<td>September to October 2014</td>
</tr>
</tbody>
</table>

7. FUNDING

This study is a low-cost research project. The student will be able to meet the running expenses which include stationary and printing. Courses organised by the university to help with data analysis and writing up of the thesis will be attended by the student.

In the majority of cases presenting with acute/sub-acute ACS it is routine practice to request baseline biomarkers pre-and post-procedure. However, those patients presenting from home for an elective PCI; a baseline cardiac enzyme is not part of routine care. The costs for these biomarker levels pre-procedure will be incurred by the department of cardiology.
8. REFERENCES


29. Cuculi F, Lim CC, Banning AP. Periprocedural myocardial injury during elective percutaneous coronary intervention: is it important and how can it be prevented? *Heart* 2010; 96(10): 736-40.

Hello my name is Dr Tsabedze I am a Masters student specialising in cardiology. As part of my degree requirements I have to do a research report that involves a review of the hospital files for patients with the condition I am investigating. I am asking for your permission to include your file in this research study. You have been selected because your blood vessels supplying blood to your heart are blocked and you are scheduled to undergo a procedure to open up these arteries. I would like to review your file to see whether there were any procedure related complications. I would also look at what risk factors you may have had that could have suggested to us that you are likely to have a complication.

What is the study about?
I am planning on reviewing a minimum of 100 patient files. I will not interfere with the patients’ routine care in the ward. I will assess file data to ascertain what form of intervention was done and whether a complication occurred or not. The following information will be retrieved from your file: Age; gender; ethnicity; other illnesses you may have; the clinical presentation; concomitant medications used; angiographic lesion factors; procedural factors; baseline and post procedure cardiac biomarkers; as well as duration of hospital stay.

Why is the study being done?
This study will enable us to define how many patients who undergo coronary intervention develop a complication in our unit. We will be able to identify key risk factors relevant to our unit associated with developing coronary intervention complications. We will be able to compare our incidence rate of procedural complications to other centers local and international. With this information we can then try and find ways of reducing the risk of procedural complications in our unit and for other centers.

Please note:
There are no risks involved in participating in this study as we are only reviewing data from your file. The decision for us to use your file is voluntary. Information obtained will be confidential and that which is reported will not identify you as a participant in this study. There are no reimbursements for allowing us to use your file data and there will be no monetary expenses incurred by you either. We will not change your management strategy we are simply reviewing your file records. This study has been approved by the Human Research Ethics Committee (Medical) of the University of the Witwatersrand, Johannesburg. Their contact details are: Tel. 011 717-1234, Fax no. 011 717 1265. E-mail: anisa.keshav@wits.ac.za.

I have read this document and fully understand its contents.
I ___________________________ grant permission for my file to be used in this study.

Sign: ____________________________  Witness: Dr N. Tsabedze
Date: ____________________________  Date: ______________
PERIPROCEDURAL MYOCARDIAL INFARCTION FOLLOWING PERCUTANEOUS CORONARY INTERVENTION AT CHARLOTTE MAXEKE JOHANNESBURG ACADEMIC HOSPITAL.

DATA COLLECTION SHEET:

Date of data collection: 

Demographic Data:

Age at time of PCI (years) 

Gender (male = 1; female = 2) 

Self Identified Ethnic Group ( African = 1; Indian = 2; White =3; Coloured =4)

Patient Risk Factors

Co-morbidities:

( Diabetes=1; Hypertension =2; CKD=3; HIV=4; PVD=5; Dyslipidaemia=6; Family Hx=7; Previous MI=8; Smoking=9 Other=10; None = 11)

Clinical Presentation

New York Heart Association Class 

Acute Coronary Syndrome:

STEMI=1; NSTEMI=2 

Non ACS Symptoms:

( Asymptomatic=1; Stable angina = 2; Unstable angina = 3)

TIMI Score:
<table>
<thead>
<tr>
<th>Test</th>
<th>Baseline</th>
<th>Post PCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-reactive protein on admission:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White cell count on admission:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Cholesterol (mmol/l)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL Cholesterol (mmol/l)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum Creatinine (umol/l)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatine Kinase MB:</td>
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<td></td>
</tr>
<tr>
<td>Baseline value (Pre-PCI)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Value at 16 to 24hrs Post PCI</td>
<td></td>
<td></td>
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<tr>
<td>Troponin - I:</td>
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<tr>
<td>Baseline value (Pre - PCI)</td>
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<td></td>
</tr>
<tr>
<td>Value at 16 to 24hrs Post PCI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Ventricular Ejection Fraction (%)</td>
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<tr>
<td>Pre-PCI Mean Arterial Pressure(mmhg)</td>
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<tr>
<td>Concomitant Medications:</td>
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</tr>
<tr>
<td>(B-blockers = 1; ACE inhibitors = 2; Statins = 3; Calcium antagonists = 4; ATII antagonists = 5; Prior use of thrombolytics =6; None = 7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angiographic Lesion Factors:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distribution of CAD:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Single vessel = 1; Double vessel = 2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Triple vessel =3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vessel Treated:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Left main = 1; LAD = 2; LCx =3; RCA = 4; Other = 5)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Lesion Site:
(Osteal = 1; Proximal = 2; Midvessel = 3; Distal = 4)

Lesion Complexity:
(A = 1; B = 2; C = 3)

Syntax Score:

Procedural Factors:

Duration of procedure (minutes):

Complications:
(Major dissection / perforation = 1;
Slow / no reflow = 2;
Significant spasm = 3;
High thrombus burden = 4;
Side branch closure / compromise = 5;
Distal embolisation = 6; None = 7)

Stent Choice:
(Drug eluting stent = 1;
Drug eluting ballon = 2;
Bare metal stent = 3;
Other = 4)

Total Stent Length (mm)

Total Volume of Contrast Used (ml)

PCI Success:
TIMI III Flow & good angiographic result = 1;
Other = 2)

Duration of Hospital Stay (days) :
APPENDIX B
UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG
Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49  Dr Nqoba I Tsabadze

CLEARANCE CERTIFICATE  M121055
PROJECT
Periprocedural Myocardial Infarction following Percutaneous Coronary Intervention at CM Johannesburg Academic Hospital

INVESTIGATORS
Dr Nqoba I Tsabadze.

DEPARTMENT
Division of Cardiology

DATE CONSIDERED
26/10/2012

DECISION OF THE COMMITTEE*
Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE  26/10/2012  CHAIRPERSON  (Professor PE Cleaton-Jones)

*Guidelines for written ‘informed consent’ attached where applicable
cc:  Supervisor:  Prof Pravin Manga

DECLARATION OF INVESTIGATOR(S)
To be completed in duplicate and ONE COPY returned to the Secretary at Room 10004, 10th Floor, Senate House, University.
I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. I agree to a completion of a yearly progress report.

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES...