IN-HOSPITAL MORTALITY OUTCOMES OF ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

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Johannesburg, April 2023

DECLARATION

Student's contribution to article(s) and agreement of co-author(s) I, Lindokuhle Wiseman Ndaba, student number 533584, declare that this research report is my work and that I contributed adequately towards research findings published in the article(s) stated below which are included in my research report.

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4	Nqoba Tsabedze		25/03/23

Comments by primary supervisor:

Dr Ndaba has worked hard and dilligently on his Research Report. This real world data reporting on Inhospital STEMI mortality in Johannesburg will help guide future research in this area.

At the time of submission, the manuscript was under review for publication.

DEDICATION

To my beloved wife, Boitumelo Ndaba, your unwavering support and love have been the bedrock of my journey. Your belief in me, even during the most challenging times, has been a constant source of strength. Your sacrifices and encouragement have propelled me forward, and I am forever grateful for having you by my side. This achievement is as much yours as it is mine, and I dedicate it to our shared dreams and the beautiful life we continue to build together.

To my brothers, Andile and Sphelele Ndaba, we have reached a milestone in our lives. The bond we share and the unbreakable support we offer each other have been pivotal in shaping who we are today. As we celebrate this accomplishment, I am reminded of the countless times we stood together, facing life's trials with resilience and unity. I am proud to call you my brothers, and I dedicate this achievement to our enduring camaraderie and the exciting adventures that lie ahead.

In this moment of celebration, I recognize the profound impact of my loved ones in shaping the person I have become. With heartfelt gratitude, I honor the love, encouragement, and unwavering belief bestowed upon me by my wife and brothers. Your presence in my life has made all the difference, and for that, I am eternally thankful.

ACKNOWLEDGEMENTS

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To my dear family and friends, your unwavering support humbles me. Your encouragement and belief in my abilities have been a driving force behind my achievements.

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This project's completion would not have been possible without the collective contributions of each individual mentioned, and I am forever grateful for their role in this transformative journey.

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Retrospective Study

In-hospital Mortality Outcomes of ST-segment Elevation Myocardial Infarction in Johannesburg, South Africa

Lindokuhle Ndaba, Arthur Mutyaba, Dineo Mpanya, Nqoba Tsabedze

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Author contributions: Ndaba L conceptualised, designed and performed the research and wrote the paper; Mutyaba A conceptualised, edited, and supervised the research project; Mpanya D contributed to the review of the manuscript, statistical analysis and validated the results; Tsabedze N conceptualised, edited and supervised the research study.

Supported by: No supporting grant.

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Abstract

BACKGROUND

ST-segment elevation myocardial infarction (STEMI) is a common malignant complication of ischaemic heart disease. Despite optimal treatment, STEMI is often associated with significant morbidity and mortality. In high-income countries (HICs), STEMI-related morbidity and mortality incidence have decreased. However, in sub-Saharan Africa (SSA), there is an increasing burden of non-communicable diseases driven by atherosclerotic cardiovascular disease. Similarly, the incidence of STEMI is increasing. However, despite the growing burden of STEMI in SSA, there is still a paucity of data reporting on the regional morbidity and mortality associated with this disease.

AIM

Hence, this study aims to provide a comprehensive description of the clinical profiles, determine the in-hospital all-cause mortality rate, and identify predictors of mortality for STEMI patients treated at the Division of Cardiology in Charlotte Maxeke Johannesburg Academic Hospital (CMJAH) in South Africa.

METHODS

We conducted a retrospective review of the medical records of 815 consecutive patients diagnosed with ST-elevation myocardial infarction (STEMI), with confirmatory angiography findings, between January 1, 2015, and December 31, 2019. All participants underwent diagnostic coronary angiography, with or without percutaneous coronary intervention (PCI). As our institution is a PCI-capable centre, all patients underwent angiography either during the index admission or at a later date, and this removed ambiguity regarding the diagnosis of STEMI. The study

included patients who met the third and fourth universal definitions of myocardial infarction.

Data on demographics, clinical presentation, electrocardiogram (ECG) changes, biochemical parameters, coronary angiographic findings, coronary interventions, STEMI-related complications, and ongoing medical management were extracted from these sources. We compared socio-demographic characteristics, comorbidities, clinical parameters, biochemistry investigation findings, treatment, and complications between the patients who survived in-hospital and those who died. To identify predictors of in-hospital all-cause mortality in patients with STEMI, we developed a multivariable logistic regression model.

RESULTS

Among 677 participants with a mean age of 55.5 ± 11.3 years, 533 (78.7%) were males. The in-hospital all-cause mortality rate was 6.2%. Co-morbidities in STEMI patients included smoking (56.1%), hypertension (52.8%), dyslipidaemia (40.0%), a family history of coronary artery disease (32.7%), and diabetes mellitus (28.3%). The median duration from the onset of chest pain to arrival at the catheterisation laboratory was two days [interquartile range (IQR): 1-3] for survivors and one day (IQR: 0-2) for non-survivors (p=0.010). Pharmaco-invasive treatment was the pre-dominant management strategy, with 61.4% of patients receiving thrombolysis and 53.9% undergoing PCI. On the age and sex-adjusted multivariable logistic regression model, the use of inotropes (OR: 4.04; CI: 1.14-14.34, p=0.031) and an increase in heart rate (OR: 1.02; CI: 1.01-1.04, p=0.010) independently predicted in-hospital all-cause mortality, while thrombolytic therapy (OR: 0.27; CI: 0.10-0.75, p=0.012), and PCI to the right coronary artery (OR: 0.03; CI: 0.00-0.46, p=0.011) reduced the risk of all-cause mortality.

CONCLUSION

Our study's all-cause mortality rate among STEMI patients is comparable to rates reported in other countries globally.

Keywords: Acute coronary syndrome; Cardiovascular epidemiology, mortality; Percutaneous coronary intervention; ST- segment elevation myocardial infarction; Sub-Saharan Africa

Core tip: In this retrospective study conducted at a single tertiary centre in Johannesburg, South Africa, we present contemporary data revealing a STEMI mortality rate of 6.2%. Notably, we identified the use of inotropes and tachycardia as significant risk factors associated with mortality. Conversely, improved outcomes were observed with the use of thrombolytic therapy and intervention to the right coronary artery (RCA). These findings demonstrate the importance of early interventions and targeted treatments. Furthermore, the mortality rate observed in our study aligns with rates reported in other global institutions, contributing valuable insights to the broader understanding of STEMI outcomes worldwide.

INTRODUCTION

In high-income countries (HICs), there has been a notable decrease in the mortality rate associated with ST-segment elevation myocardial infarction (STEMI) (1). However, sub-Saharan Africa (SSA) has experienced a significant surge in risk factors related to STEMI, such as hypertension, smoking, dyslipidemia, and diabetes. The increase in the prevalence of risk factors for cardiovascular diseases (CVD) can be attributed to urbanisation, dietary and lifestyle changes, and an aging population (2, 3). Consequently, ischemic heart disease (IHD), cerebrovascular disease, and hypertensive heart disease have emerged as the leading causes of cardiovascular mortality in SSA (3). The high burden of non-communicable diseases in SSA has put immense strain on an already overwhelmed and under-equipped healthcare infrastructure.

In SSA, the mortality rate associated with STEMI exhibits considerable variation and can be as high as 24.5% (2). Timely access to coronary revascularisation strategies is a critical determinant of mortality outcomes in SSA among patients with acute coronary syndromes (ACS) (2). However, several factors contribute to the mortality burden, including lack of patient education, delayed health-seeking behaviour, unequal health systems, inappropriate triage, incomplete revascularisation, and limited access to

primary percutaneous coronary intervention (PCI) and coronary artery bypass graft surgery (2).

A review of the South African arm of the Acute Coronary Events (ACCESS) registry, a multinational survey of current management strategies of patients hospitalised with ACS in 19 African countries, underestimated the in-hospital mortality rate, due to a small representation of South African patients in the registry, with only 642 out of 12,068 patients, and 253 individuals with confirmed STEMI (4). There is limited data reporting outcomes in patients with STEMI residing in SSA, particularly in South Africa. This study aimed to describe risk factors and the clinical presentation of patients hospitalised with STEMI in a tertiary academic hospital in Johannesburg, South Africa. Furthermore, we aimed to determine the in-hospital all-cause mortality rate and identify predictors of all-cause mortality among STEMI patients.

MATERIALS AND METHODS

Study Design

We conducted a cross-sectional retrospective single-centre study of consecutive patients diagnosed with STEMI, with concomitant angiography, between 01 January 2015 and 31 December 2019. The study was conducted at the Charlotte Maxeke Johannesburg Academic Hospital (CMJAH) in Johannesburg, South Africa, which is a state-owned hospital that serves individuals without medical insurance. The CMJAH is a state-owned academic tertiary hospital in central Johannesburg, forming part of the University of the Witwatersrand academic cluster of teaching hospitals. Patients 18 years and older who met the inclusion criteria for STEMI, as per the third and fourth Universal Definitions of Myocardial Infarction, were included in the study (5). An overlap in the definitions occurred because the third universal definition was released in 2012, while the fourth universal definition was released in 2018. Consequently, records from 2015 to 2018 utilised the third universal definition, whereas records from 2018 to 2019 used the fourth universal definition.

Patients with incomplete medical records (pertinent missing data such as gender, ACS presentation, electrocardiogram (ECG) findings, laboratory results, intervention and clinical outcome), duplicate medical records, a working diagnosis of non-ST elevation

acute coronary syndromes (NSTACS), alternate diagnoses such as acute peri-myocarditis, takotsubo cardiomyopathy and trauma-related myocardial infarction were excluded from the final analysis. Premature coronary artery disease in our study referred to individuals under the age of 50 years old. Ethics approval was obtained from the University of the Witwatersrand Human Research Ethics Committee (Clearance Certificate Number: M201095) and relevant hospital authorities. The authors adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Data Collection

Inpatient medical records for 815 patients were retrieved. A thorough verification process was conducted, which involved reviewing the medical records, archived coronary angiogram reports, coronary angiogram suite admission records, and Cardiac Intensive Care Unit (CICU) admission records. Patients with incomplete medical records, records, non-ST-segment elevation acute coronary syndrome (NSTACS), and alternative diagnoses were excluded from the final study analysis. As a result, a total of 677 patients with ST-elevation myocardial infarction (STEMI) were included in the study (Figure 1).

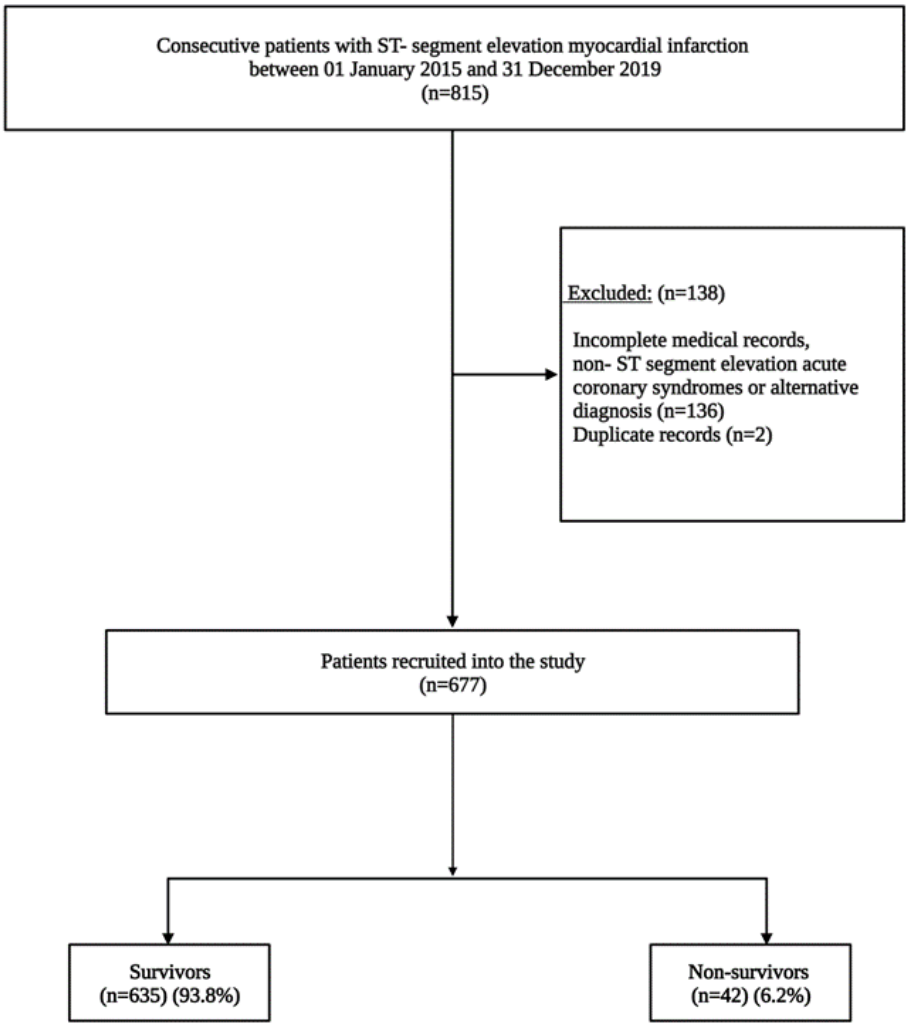

Figure 1 Flow chart outlining enrollment of patients into the study. STEMI: ST-elevation myo-cardial infarction

To ensure the validity and reliability of the data abstraction process, specific measures were implemented. First, the data abstractor underwent comprehensive training in the standardized methods of data abstraction from medical records. This training encompassed familiarizing the abstractor with the study objectives, data collection instruments, and the specific variables to be extracted.

To maintain consistency and quality control, abstractor monitoring was conducted by experienced researchers. This involved periodic reviews of a subset of medical records abstracted by the abstractor to identify any discrepancies or inconsistencies. Feedback and clarification sessions were conducted as necessary to ensure the accuracy and uniformity of the data abstraction process. No blinding was performed due to only one data collector being responsible for data collection and the individual collecting the data was aware of the study hypotheses and outcomes of interest.

Interrater reliability was assessed periodic re-abstraction of a subset of medical records for cross-validation and ensuring adherence to standardized data abstraction protocols.

Regarding missing, duplicate, and conflicting data entries, a standardized protocol was followed. Missing data were noted, and efforts were made to retrieve the necessary information from alternative sources or by contacting healthcare providers involved in the patients' care. Duplicate entries were identified by cross-referencing patient identifiers, and only a single entry for each patient was retained. Conflicting data entries were carefully reviewed, and efforts were made to reconcile the discrepancies by referring back to the primary medical records or seeking additional clarification from healthcare providers.

The collected data included various variables such as demographics (age, gender, and ethnicity), co-morbidities, smoking and alcohol use, clinical presentation (onset of chest pain, time from onset of chest pain to first medical contact and catheterization), admission ECG parameters (rhythm, rate, ST-segment elevation and depression, and bundle branch blocks), clinical parameters (blood pressure, New York Heart Association functional class, and Killip class), biochemical parameters (cardiac biomarkers, renal function, lipogram, and hemoglobin), coronary angiography findings (dominant vessel, distribution of disease on angiography, and the number of lesions), coronary intervention details (adjunct medical therapy received, thrombolytic therapy, and percutaneous coronary intervention), post-intervention complications (arrhythmias and hemodynamic instability), medications, and in-hospital all-cause mortality.

All the data were captured and recorded in the Research Electronic Data Capture (REDCap) database.

Statistical Analysis

Frequencies and percentages were used to summarise categorical variables. The mean and standard deviation (SD) were used to summarise normally distributed continuous data, and the median and interquartile ranges (IQR) were used for continuous variables with a non-normal distribution. We compared demographic and clinical parameters between survivors and non-survivors. For continuous variables, the Student's t-test was used to compare means for normally distributed variables. The Wilcoxon rank-sum test was used to compare medians for data with a non-normal distribution. Pearson's Chi-square test was used to compare categorical variables. Univariable and multivariable logistic regression analyses were used to determine the predictors of in-hospital all-cause mortality. The odds ratios in the multivariable model were adjusted for confounders such as age and gender. After conducting a Pearson's Chi-square test, Student's t-test or a Wilcoxon rank-sum test variables with a p-value < 0.1 were considered for further exploration in the univariable logistic regression model. We further included all variables from the univariable logistic regression model with a p-value less than 0.05 in the multivariable logistic regression model. A detailed explanation of how the multivariable logistic regression model was created is attached as a supplementary file. We presented the crude and adjusted odds ratios with their 95% confidence intervals (95% CI). Statistical significance was considered at a p-value < 0.05. Analysis was performed using Stata version 17 (StataCorp Ltd, College Station, TX).

RESULTS

The final study population comprised 677 patients, of which 533 (78.8%) were males. The mean age was 55.5 ± 11.3 years. Overall, 319 (47.1%) patients were Caucasians, 167 (24.7%) were Black, 143 (21.1%) were Indian or Asian, and 38 (5.6%) were of Mixed Ancestry. Prominent co-morbidities were smoking (56.1%), hypertension (52.8%), dyslipidaemia (40.0%) and diabetes mellitus (28.3%), while previous coronary artery disease was documented in only 3.1% of the study population, and 222 (32.8%) patients had a family history of heart disease (Figure 2).

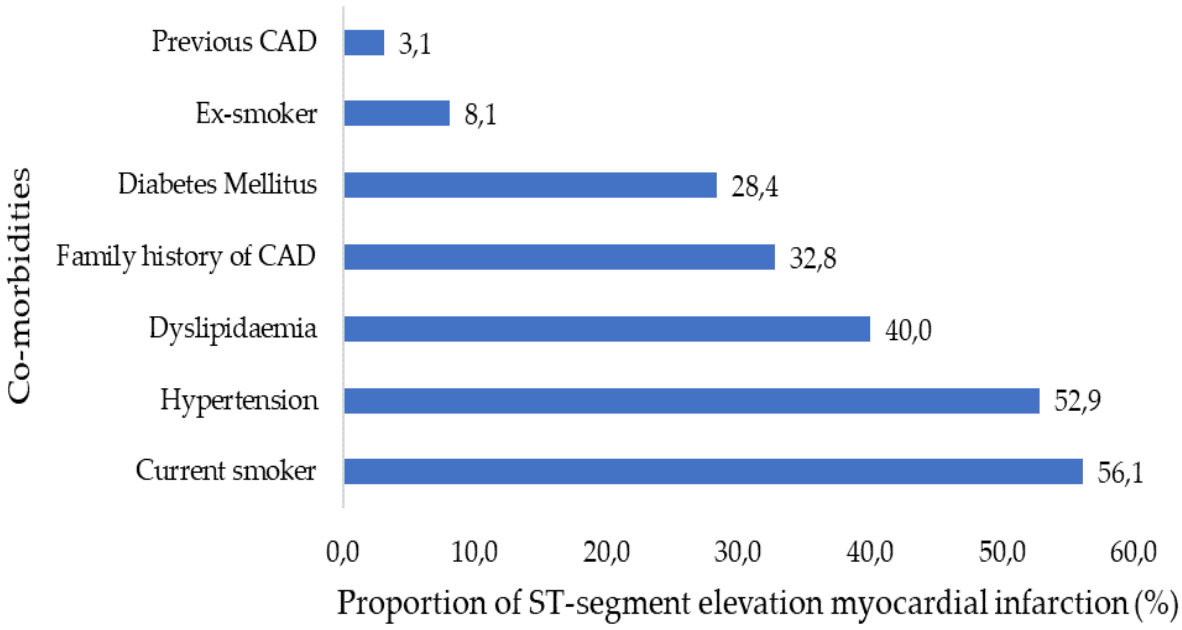

Figure 2 Co-morbidities for ST-elevation myocardial infarction. CAD: coronary artery disease.

Thirty-four patients had human immunodeficiency virus (5.02%), three patients (0.4%) had a documented history of excessive alcohol intake, and five (0.7%) had a history of recreational drug use. During the study period, 42 STEMI patients died, and the in-hospital all-cause mortality rate was 6.2%. Non-survivors were older, with a mean age of 60.5 ± 14.1 years, compared to survivors with a mean age of 55.2 ± 11.1 years (p<0.001). The median time in days from the onset of chest pain to the catheterisation laboratory was shorter among non-survivors [1 (IQR: 0-2) vs 2 (IQR: 1-3) days, p=0.01]. During hospitalisation, non-survivors had lower blood pressure than survivors, with a systolic blood pressure of 104 vs 126 mmHg (p<0.001) and a diastolic blood pressure of 68 vs 78 mmHg (p<0.001). The heart rate was higher in non-survivors (99 vs 83 beats per minute, p<0.001) (Table 1).

Table 1: Baseline demographic and clinical parameters in patients with ST-segmentelevation myocardial infarction at the time of hospitalisation

Variables	**All patients n=677**	**All-cause mortality**		**p-value**
		No (n = 635) (93.8%)	**Yes (n = 42) (6.2%)**	
Age, years	55.54 ± 11.39	55.21 ± 11.11	60.54 ± 14.11	<0.001
Male	533 (78.73)	503 (79.21)	30 (71.43)	0.233
Co-morbidities				
Hypertension	358 (52.88)	335 (52.76)	23 (54.76)	0.801
Diabetes Mellitus	192 (28.36)	179 (28.19)	13 (30.95)	0.700
Dyslipidaemia	271 (40.03)	255 (40.16)	16 (38.10)	0.792
Obesity	153 (22.60)	144 (22.68)	9 (21.43)	0.851
Chronic kidney disease	17 (2.51)	17 (2.68)	0 (0.00)	0.283

Previous CAD	21 (3.10)	20 (3.15)	1 (2.38)	0.781
HIV	34 (5.02)	33(5.20)	1(2.38)	0.418
Smoking				
Current smoker	380 (56.13)	362 (57.01)	18 (42.86)	0.073
Ex-smoker	55 (8.12)	49 (7.72)	6 (14.29)	0.131
Vital signs				
Systolic BP (mmHg)	124.41 ± 26.15	125.77 ± 25.09	103.46 ± 32.94	<0.001
Diastolic BP(mmHg)	78.65 ± 18.49	79.36 ± 18.12	67.76 ± 20.92	<0.001
Heart rate	84.38 ± 22.26	83.42 ± 20.37	98.83 ± 39.06	<0.001
NYHA class				
1	601 (88.77)	583 (91.81)	18 (42.86)	<0.001
2	37 (5.47)	30 (4.72)	7 (16.67)	0.001
3	17 (2.51)	14 (2.20)	3 (7.14)	0.048
4	22 (3.25)	8 (1.26)	14 (33.33)	<0.001
Killip class				
1	576 (85.08)	560 (88.19)	16 (38.10)	<0.001
2	30 (4.43)	26 (4.09)	4 (9.52)	0.098
3	24 (3.55)	17 (2.68)	7 (16.67)	<0.001
4	47 (6.94)	32 (5.04)	15 (35.71)	<0.001
Biochemical Parameters				

Troponin T (ng/l)	3556 (1495-7189)	3444 (1389-6893)	6774 (2732-10000)	0.004
CK MB mass (ug/l)	82.67 (20.17-239.1)	72.35 (19.38-203.6)	268 (207.2-268)	0.013
Haemoglobin (g/dl)	14.34 ± 2.20	14.34 ± 2.18	14.32 ± 2.61	0.972
HbA1C (%)	8.7 (7.3-10.5)	8.7 (7.4-10.6)	7.9 (7.0-8.9)	0.171
Sodium (mmol/L)	138 ± 9.12	139.21 ± 7.87	135.29 ± 19.80	0.007
Potassium (mmol/L)	4.2 (3.9-4.6)	4.2 (3.9-4.6)	4.45 (4.0-5.2)	0.021
Urea (mmol/L)	5.6 (4.4-7.4)	5.5 (4.4-7.1)	7.9 (6.0-11.7)	<0.001
Creatinine (umol/L)	87 (73-107)	86 (72.5-104)	115 (82-149)	<0.001
eGFR (ml/min/1.73m^2)	78.35 ± 27.21	79.78 ± 26.52	56.64 ± 28.62	<0.001
Electrocardiogram findings				
Sinus rhythm	610 (90.10)	581 (91.50)	29 (69.05)	<0.001
Atrial fibrillation	12 (1.77)	10 (1.57)	2 (4.76)	0.130
Atrial flutter	3 (0.44)	3 (0.47)	0 (0.00)	0.655
Ventricular tachycardia	7 (1.03)	6 (0.94)	1 (2.38)	0.373
Ventricular fibrillation	5 (0.74)	3 (0.47)	2 (4.76)	0.002
Heart block	22 (3.25)	16 (2.52)	6 (14.29)	<0.001
Left bundle branch block	19 (2.81)	18 (2.83)	1 (2.38)	0.186
Right bundle branch block	24 (3.55)	20 (3.15)	4 (9.52)	0.186

BP: blood pressure, CAD: coronary artery disease, CK: creatine kinase, ECG: electrocardiogram, eGFR: estimated glomerular fil tration rate, HbA1c: glycated haemoglobin, NYHA: New York Heart Association

The left anterior descending artery (LAD) lesions were found in 78.6% of non-survivors (p=0.009). Triple vessel disease was significantly associated with poor clinical outcomes, noted in 30.9% of non-survivors and 12.3% of survivors (p=0.001). The rest of the diagnostic coronary angiography parameters, PCI, and post-intervention complications are depicted in Table 2.

Table 2 Coronary angiography findings and interventions in patients with ST-segment elevation myocardial infarction

Variables	All patients n=677	All-cause mortality		p-value
		No (n = 635) (93.8%)	Yes (n = 42) (6.2%)	
Vessels involved				
Right coronary artery	351 (51.85)	330 (51.97)	21 (50.00)	0.850
Left main coronary artery	14 (2.07)	12 (1.89)	2 (4.76)	0.205
Left circumflex artery	156 (23.04)	142 (22.36)	14 (33.33)	0.102
Left anterior descending artery	403 (59.53)	370 (58.27)	33 (78.57)	0.009
Obtuse marginal/Ramus	29 (4.28)	27 (4.25)	2 (4.76)	0.874
First diagonal branch	13 (1.92)	12 (1.89)	1 (2.38)	0.822
Distribution of disease on angiography				
Single vessel disease	390 (57.61)	369 (58.11)	21 (50.00)	0.303
Double vessel disease	143 (21.12)	137 (21.57)	6 (14.29)	0.262
Triple vessel disease	91 (13.44)	78 (12.28)	13 (30.95)	0.001
No lesion*	20 (2.95)	20 (3.15)	0 (0.00)	0.243

Intervention				
Aspirin	673 (99.41)	631 (99.37)	42 (100.00)	0.606
Clopidogrel	673 (99.41)	631 (99.37)	42 (100.00)	0.606
Clexane	674 (99.56)	632 (99.53)	42 (100.00)	0.655
Thrombolysis	416 (61.45)	397 (62.52)	19 (45.24)	0.026
Inotropes				
Dobutamine	49 (7.24)	30 (4.72)	19 (45.24)	<0.001
Adrenaline	17 (2.51)	5 (0.79)	12 (28.57)	<0.001
Phenylephrine	6 (0.89)	1 (0.16)	5 (11.90)	<0.001
Pacing	20 (2.95)	13 (2.05)	7 (16.67)	<0.001
Percutaneous coronary intervention				
Percutaneous coronary intervention	365 (53.91)	347 (54.65)	18 (42.86)	0.125
Left main coronary artery	1 (0.15)	1 (0.16)	0 (0.00)	0.797
Left anterior descending artery	187 (27.62)	172 (27.09)	15 (35.71)	0.226
Left circumflex artery	40 (5.91)	36 (5.67)	4 (9.52)	0.305
Right coronary artery	149 (22.01)	146 (22.99)	3 (7.14)	0.016
Obtuse marginal artery	1 (0.15)	1 (0.16)	0 (0.00)	0.797
Obtuse marginal/Ramus	8 (1.18)	8 (1.26)	0 (0.00)	0.464

In addition, 50.0% of the non-survivors required inotropes (p<0.001), and a further 16.6% required cardiac pacing (p<0.001). Post-intervention, 39 (5.8%) patients demonstrated haemodynamic instability, and 29 (74.3%) of these patients died (p<0.001). Al-so, 11 patients (1.6%) had ventricular fibrillation, 10 (1.5%) had a ventricular tachycardia, and 9 (1.3%) had atrial fibrillation. ST-segment elevation myocardial infarction complicated by atrial fibrillation, ventricular fibrillation, sustained and non-sustained ventricular tachycardia, and complete heart block (CHB) were significantly associated with mortality (p<0.001). The median duration of hospitalisation was three days (IQR: 2-6). The median time to death among non-survivors was 2.5 days (IQR: 1-9). Upon discharge from the hospital, 667 (98.5%) STEMI patients were prescribed oral angiotensin receptor blocker antagonists, 624 (92.2%) were on oral dual antiplatelet therapy, 517 (76.4%) were on beta-blockers, and 436 (64.4%) were on angiotensin-converting enzyme inhibitors. The pre-scription of mineralocorticoid receptor antagonists was uncommon, with only 67 (9.9%) patients on this oral medication. On the age and sex-adjusted multivariable logistic regression model, the use of inotropes (OR: 4.04; CI: 1.14-14.34, p=0.031), an in-crease in heart rate (OR: 1.02; CI: 1.01-1.04, p=0.010) were associated with an increased risk of all-cause mortality, while thrombolytic therapy (OR: 0.27; CI: 0.10-0.75, p=0.012), and PCI to the right coronary artery (RCA) (OR: 0.03; CI: 0.00-0.46, p=0.011) reduced the risk of all-cause mortality. (Table 3).

Table 3: Univariable and multivariable logistic regression analysis of predictors of in-hospital all-cause mortality in patients with ST-segment elevation myocardial infarction

	Univariable Logistic Regression		**Multivariable Logistic Regression**			
Variable	**OR (95% CI)**	**p- value**	**Crude OR (95%CI)**	**p-value**	**Adjusted OR* (95% CI)**	**p-value**
Age (years)	1.04 (1.01-1.07)	0.004			1.04 (1.00-1.09)	0.125
Systolic BP	0.96 (0.95-	<0.001	97 (0.94-1.00)		0.96 (0.93-	

	0.98)			0.065	1.00)	0.035
Diastolic BP	0.96 (0.94-	<0.001	1.00 (0.96-		1.01 (0.97-	
	0.98)		1.06)	0.833	1.07)	0.569
Troponin	1.00 (1.00-	0.007	1.00 (1.00-		1.00 (0.99-	
	1.00)		1.00)	0.386	1.00)	0.426
Sodium	0.98 (0.96-	0.030	0.99 (0.96-		0.99 (0.96-	
	1.00)		1.02)	0.387	1.02)	0.436
Potassium	1.03 (1.00-	0.038	1.03 (0.99-		1.03 (1.00-	
	1.06)		1.07)	0.158	1.07)	0.149
Urea	1.04 (1.00-	0.014	1.02 (0.98-	0.267	1.02 (0.98-	
	1.07)		1.07)		1.07)	0.303
Creatinine	1.01 (1.00-	0.001	1.00 (1.00-		1.00 (1.00-	
	1.01)		1.01)	0.539	1.01)	0.254
eGFR	0.96 (0.95-	<0.001	0.96 (0.94-		0.97 (0.94-	
	0.98)		0.99)	0.013	1.00)	0.049
Tachycardia	1.02 (1.01-	<0.001	1.02 (1.00-		1.02 (1.01-	
	1.04)		1.04)	0.012	1.04)	0.010
Thrombolytic	0.50 (0.26-	0.028	0.26 (0.09-		0.27 (0.10-	
therapy	0.93)		0.71)	0.009	0.75)	0.012
Inotropes	19.48 (9.63-	<0.001	4.15 (1.16-	0.028	4.04 (1.14-	
	39.41)		14.77)		14.34)	0.031
PCI to RCA	0.26 (0.08-	0.025	0.04 (0.00-		0.03 (0.00-	
	0.85)		0.48)	0.012	0.46)	0.011

BP: blood pressure, CI: confidence interval, eGFR: estimated glomerular filtration rate, OR: odds ratio, PCI: Percutaneous coronary intervention, RCA: Right coronary artery. *Age and sex-adjusted odds ratio.

DISCUSSION

In this study, we retrospectively reviewed medical records of 677 patients diagnosed with ST-segment elevation myocardial infarction (STEMI). We found an in-hospital all-cause mortality rate of 6.2%. Ischemic heart disease (IHD), cerebrovascular disease, and hypertensive heart disease are SSA's most common causes of cardiovascular mortality (3). Early studies showed a low incidence rate of IHD in SSA (6, 7). However, a steady rise was noted in the early 2000s (8, 9). The rising number of patients with IHD could be accounted for by the rise in urbanisation, increased access to processed food and a sedentary lifestyle. The previously reported lower incidence rate of IHD could be explained by underdiagnosis and paucity of research data reporting outcomes in STEMI patients residing in SSA, particularly those treated in state-owned hospitals. Limited access to diagnostic tools such as biomarkers reflecting myocardial injury, ECGs, and coronary angiography may result in the underdiagnosis of STEMI (10). Furthermore, the incidence and prevalence rates of IHD reported in SSA in older research studies may be inaccurate, partly because of patients demising before arrival in hospitals equipped with catheterisation laboratories (11).

Smoking and hypertension were the most common risk factors in our study population, reported in 56.1% and 52.8% of STEMI patients. These were closely followed by dyslipidaemia in 40.0%, a family history of heart disease in 32.7%, diabetes mellitus in 28.3%, and obesity in 22.6%. The commonly reported modifiable risk factors for CVD include smoking, diabetes, hypertension, obesity, hyperlipidemia, physical inactivity, and an unhealthy diet, which are becoming more prevalent in SSA. However, the true burden of risk factors for CAD has not been clearly defined due to fewer research studies performed in our region and deficient systems for data collection (12). The proportion of smokers residing in 29 SSA countries (South Africa not included in the analysis) is between 4.6-25.8% (3), and the prevalence rate of hypertension is higher among individuals residing in urban areas, estimated at 40% (13). In comparison, their counterparts in rural areas had a lower prevalence rate of 20% (13). In 2014, diabetes mellitus had a lower prevalence rate of 7.1% among individuals residing in Africa (12). Also, in a meta-analysis involving 117

studies with 294,063 participants, the pooled prevalence of dyslipidaemia in the general adult population residing in Africa was 25.5% (95% CI: 20.0-31.4) (14). Guthold et al. conducted a survey involving 57,038 individuals from 22 African countries and found that at least 24.3% and 16.2% of females and males did not meet the World Health Organisation's physical activity recommendations (15). The age-standardised prevalence of obesity among men and women 20 years and older in South Africa is 14.7% and 44.6%, respectively (16). There is growing evidence that primary prevention of these risk factors within SSA may be a reasonable step to reduce the growing burden of CVD (17). Other socio-economic factors identified as major psychological stressors contributing to an unhealthy diet, low physical activity, obesity, increased alcohol consumption, and smoking include poor housing, low income, poor sanitation and limited access to healthcare facilities (18). Also, most of the population in SSA resides in overpopulated informal settlements, an environment that is not optimal for establishing healthy behaviours.

The low number of patients with a prior history of angina and the high proportion of patients with double or triple multivessel CAD could be attributed to various factors. One possibility is that angina may be underreported or undiagnosed in some patients, leading to a lower documented prevalence. Certain condition populations are also known to have silent cardiovascular disease, namely the elderly, females, and diabetic patients. Additionally, other risk factors such as genetics, lifestyle choices, or comorbidities could play a significant role in the development and severity of CAD, regardless of prior angina symptoms. Further research and analysis would be necessary to explore these potential explanations.

Our study found an in-hospital all-cause mortality rate of 6.2% among STEMI patients. The Kerala Acute Coronary Syndrome (ACS) Registry in India reported a mortality rate of 8.2% after evaluating 25,748 patients hospitalised with acute myocardial infarction, of which 9,569 had STEMI (19). Similarly, 18,631 patients in China with acute myocardial infarction, of which 13,815 were STEMI admissions, had a mortality rate of 7.0% (20). The management of ST-segment elevation myocardial infarction (STEMI) in China, India, and South Africa demonstrates a mix of advancements and

obstacles (4). China boasts a rapidly evolving healthcare infrastructure that grants urban populations access to cutting-edge cardiac care (20). In India, a diverse healthcare landscape results in varying capacities, yet rural areas grapple with significant disparities (19). Conversely, South Africa's urban centres excel in providing well-established STEMI services, but rural regions suffer from insufficient care provisions (4). Notably, prehospital systems exhibit divergent levels of progress, with China and India displaying improvements (19,20), while South Africa encounters challenges. While major cities in these countries possess advanced technology and expertise, equitable distribution remains a concern. Disparities in healthcare financing and affordability further characterize these regions. China is actively working to curtail expenses associated with healthcare (20). To achieve more uniform STEMI care and alleviate mortality disparities, it is imperative to address these discrepancies comprehensively. The comparisons between China, India, and South Africa underscore the importance of context-specific strategies. China's urban-focused advancements, India's mixed capabilities amidst rural-urban disparities, and South Africa's urban-rural divide emphasize the need for tailored interventions. Enhancing prehospital systems, expanding access to advanced technology, and improving healthcare affordability are key steps toward achieving more equitable and effective STEMI management in these diverse healthcare landscapes.

Compared to other single-centre registries, our study demonstrated a lower mortality rate. For instance, Takagi et al. reported an in-hospital mortality rate of 9.2% in 2021 in a study consisting of 1735 participants, while Ali et al. found a mortality rate of 10% in their single-centre study involving 312 patients at Harzklinik Goslar in Germany (21).

The STEMI-related mortality in SSA is between 1.2 - 24.5% (2). This wide range in mortality rate is likely caused by the variation in the populations studied, where patients with different types of ACS presentations are included, such as STEMI, non-ST-elevation myocardial infarction (NSTEMI) and unstable angina. Also, limited access to revascularisation procedures may lead to a higher mortality rate. For instance, in a study by N'Guetta et al., the lowest mortality rate of 1.2% was observed

(22). Their study focused on patients hospitalised with ACS who underwent PCI. On the other end of the spectrum, the highest mortality rate observed in Ethiopia might have been influenced by exceptionally long treatment times, averaging approximately 96 hours. A review of the subset of data obtained from participants recruited in South Africa that are part of the ACCESS registry reported a STEMI mortality rate of 0.65% (4). The nature of the study design can explain this low value since the ACCESS registry was an observational study.

that enrolled an urban population at tertiary care facilities. Also, the enrolling centres were mainly private hospitals.

In 2017, South Africa was one of the five SSA countries with access to PCI, including Kenya, Côte d'Ivoire, Sudan, and Mauritania. South Africa had 62 PCI centres in 2017, most of which were in private hospitals (23), compared to 12 catheterisation laboratories distributed over three countries in SSA (24). This discrepancy in access to revascularisation laboratories may explain the improved mortality outcomes in South Africa compared to other countries in the SSA region. Between 1990 and 2007, most low-and-middle-income countries (LMICs) focused on combatting the burden of communicable diseases (25). Despite the growing burden of CVD, approximately 3% of global funding was allocated to non-communicable diseases (25).

The higher mortality rate in South Africa's public sector compared to the private sector could arise from selection bias, excluding patients dying before reaching Cardiac Medical Hospitals (CMH). Limited access to primary percutaneous coronary intervention (PCI), delayed presentation, and resource constraints contribute to this bias. Disparities in comorbidities and socioeconomic challenges within the public sector further impact outcomes. Addressing these issues necessitates improved STEMI care accessibility, prehospital systems, public awareness, and resource allocation. Methodological differences also explain mortality disparities seen in the ACCESS trial. The private sector's funded nature allows unhindered catheterization access and heightened symptom awareness. Effective solutions require comprehensive strategies to rectify these inequalities.

Atherosclerotic cardiovascular disease (ASCVD) is traditionally associated with advanced age, particularly in high-income countries with higher life expectancy. In contrast, our study population had a mean age of 55 years. This finding is similar to reports from other regional studies (2, 4). Of note, our study patients who demised were older than those who survived, with a mean age of 60.5 years. While our population is younger than the rest of the world, the STEMI patients in our study that demised were older in the context of life expectancy in our country.

In our study, people of Caucasian ancestry comprised 47.1% of the population studied, followed by those of African ancestry. In most LMICs, acute coronary syndromes are more prevalent among Caucasians (4). In South Africa, most individuals residing in our country are of African descent. Overall, our study findings align with the literature reporting a higher prevalence of IHD among Caucasian and Asian people, however these races are likely underrepresented because a majority receive medical treatment in the private sector. However, race did not play role in mortality outcomes.

There was a predominance of the male gender, with 79.2% of the population being male. The male gender is a non-modifiable risk factor for ASCVD. This is attributed to a higher burden of risk factors in men, with dyslipidaemia and smoking being the most prominent (26). In addition, Larsson et al. suggest that the difference in body fat distribution could also account for the higher risk in men (27). However, the Effect of Potentially Modifiable Risk Factors Associated with Myocardial Infarction (INTERHEART) case-control study found that in both males and females, abdominal obesity doubled the risk of acute myocardial infarction (28). However, after adjusting for other risk factors, such as apolipoproteins, the risk of acute myocardial infarction was substantially reduced in both genders (28).

In our study, STEMI patients who received inotropes were four times more likely to die. Patients who received inotropes most likely had underlying life-threatening conditions, such as cardiogenic shock, that warrant using inotropes. This finding is consistent with findings reported in the literature, where cardiogenic shock is associated with a 30-day mortality rate of approximately 50% due to reduced organ

perfusion, leading to critical and life-threatening organ failure (29). We also found that more than half of STEMI patients had culprit lesions in the LAD. However, the age and sex-adjusted multivariable logistic regression model showed that PCI targeting the RCA was protective and reduced the risk of in-hospital all-cause mortality. This finding suggests that successful complete revascularisation, including the RCA may positively impact patient prognosis. Also, it emphasises the importance of accurately identifying the stenosed coronary artery and performing timely interventions to achieve optimal outcomes.

The United States Global Registry of Acute Coronary Events (GRACE) registry demonstrated that heart rate is one of the eight predictors of mortality (30). In our study, non-survivors had a mean heart rate of 99 ± 39.0 beats per minute (bpm). A heart rate between 70 and 79 bpm is considered low risk, while a rate above 80 bpm is associated with a 2.2-5.3-fold increased risk of in-hospital mortality (31). Similarly, we found that an increase in the heart rate independently predicted mortality among STEMI patients. One possible explanation for this association is that tachycardia is a marker of excess sympathetic activation in response to increased stress. The higher mortality risk in STEMI patients with tachycardia emphasises the importance of promptly recognising and treating the cause of tachycardia in STEMI patients to prevent adverse outcomes.

Troponin release follows predictable kinetics, and peak levels correlate with ischemic time and the amount of at-risk myocardium (32). Additionally, troponins exhibit excellent cardiac specificity, and higher peak troponin levels have been associated with ventricular dysfunction and mortality (33, 34). In our study, non-survivors had higher levels of troponin, almost twice as high as that of survivors. However, troponin levels did not emerge as a significant predictor of mortality in the multivariable logistic regression model. Hyperglycaemia has also been reported as an important predictor of mortality (35). However, our study did not document nor analyse blood glucose levels among STEMI patients. We were able to document and analyse HbA1c levels in diabetic patients. Although fingerstick testing is routinely

performed in patients admitted to our hospital, formal random glucose testing is rarely done due to resource constraints unless there is a specific clinical indication.

The implementation of a primary PCI strategy and minimising prehospital delays in high-income countries have resulted in decreased morbidity and mortality associated with STEMI (1). In our study population, the median duration between the onset of chest pain and catheterisation was approximately two days. Among the participants that underwent coronary angiography, 53.9% received PCI. Unfortunately, we did not have access to catheterisation reports in the study and therefore, it was difficult to ascertain the rationale for intervention in specific patients. However, it is known that there are many factors that determine the decision to intervene, ranging from patient specific, procedural and prevailing evidence. In the scenario where an occluded vessel is found in a patient with ST-elevation myocardial infarction (STEMI), the decision to intervene would typically involve the consideration of several factors. The primary goal is to restore blood flow to the affected area of the heart as quickly as possible. Interventional cardiologists may assess the extent of ischemia, the location and severity of the occlusion, the presence of ongoing symptoms, and the patient's clinical stability. Based on these factors, a decision may be made to perform percutaneous coronary intervention (PCI) to open the occluded vessel using techniques such as balloon angioplasty and stent placement. However, individual patient characteristics and institutional protocols may also influence the decision-making process. However, due to resource constraints, an overwhelmed referral network, and potentially suboptimal triage systems, a pharmaco-invasive strategy is the standard of care in our clinical setting. Our study observed a long pain-to-balloon time and a reasonable survival rate, likely attributed to our institution being a tertiary referral centre. This may have led to the inclusion of patients who survived until referral, while those who died before referral to our institution were not included in the study's final analysis. Additionally, a pharmaco-invasive approach is commonly employed in our hospital, where patients are often routinely treated with thrombolytic therapy before catheterisation. Our study examined the impact of different treatment approaches on all-cause mortality, and we found that STEMI patients treated with thrombolytic agents were less likely to die. This finding supports the effectiveness of early

thrombolytic therapy in improving patient outcomes. It emphasises its value, particularly in resource-limited settings where immediate access to catheterisation laboratories may be challenging. In SSA, thrombolytic therapy is often readily available, offering similar clinical benefits to primary PCI (36). Due to its lower cost, Streptokinase is the most accessible thrombolytic agent in SSA (2, 4), although Alteplase is also used in South Africa (37). In our study population, 61.4% received thrombolytic therapy. We hypothesise that the inadequate thrombolysis rates may have resulted from limited access to thrombolytics, late presentation beyond the therapeutic time window of thrombolytic therapy, and physician inertia.

The findings of this study have important implications for clinical practice. Firstly, patients presenting with tachycardia should receive close monitoring due to the increased risk of adverse outcomes. Similarly, patients receiving inotropes should be regularly reviewed to assess their response to treatment and identify any potential complications.

Thrombolytic therapy should be administered promptly and appropriately, as it has been associated with favourable outcomes. Clinicians should consider this treatment option when appropriate, based on the patient's clinical characteristics and the presence of contraindications.

Regarding lesions in the right coronary artery (RCA), it is crucial to recognize that while they may initially be considered benign, intervention can lead to improved outcomes. Therefore, an active approach should be taken in evaluating and managing RCA lesions, considering the potential benefits of timely intervention.

These recommendations underscore the importance of tailored and vigilant management strategies for patients with specific clinical features, including tachycardia, inotrope use, thrombolytic therapy, and RCA lesions. Implementing these recommendations in clinical practice can contribute to better patient outcomes and improved overall care.

LIMITATIONS

The study had several limitations. Firstly, the retrospective nature of the study design introduced numerous problems, such as illegible documentation and incomplete medical records. Secondly, the study data were obtained from a single centre, limiting the generalizability of the findings. Electrocardiogram parameters were mainly gathered from clinical notes made by the cardiologists instead of reviewing actual ECG printouts. Thirdly, the ultimate cause of death was not routinely documented. Fourthly, there was an overlap between the use of the third and fourth universal definitions of myocardial infarction, resulting in the use of the third definition from 2015 to 2018 and the fourth definition from 2018 to 2019. We did not clearly articulate which patients which patients received complete revascularisation. Furthermore, at our institution, the primary lesion gets intervention and if the other lesions are clinically or physiologically relevant than a decision to intervene is made. Complete revascularisation is however rarely performed at the initial angiography. Unfortunately, a large portion of our patients are lost to follow up for repeat angiography. We also did not articulate, and which patients required intra-aortic balloon pumps. The need for mechanical circulatory support is a vital area of interest in STEMI and it was an oversight to not include it. We could only narrow down timelines from chest pain to presentation and catheterisation to calendar days and not hours. Due to the retrospective nature of the study, we were only able to document mortality but not the cause of mortality. Lastly, pertinent clinical parameters such as the left ventricular ejection fraction and serum glucose levels were not collected and analysed as part of the study. Despite these limitations, our study provides valuable insights into the burden of co-morbidities in STEMI patients and estimated the rate of in-hospital all-cause mortality in STEMI patients managed in a tertiary academic centre in Johannesburg, South Africa

CONCLUSIONS

In our study, the in-hospital, all-cause mortality rate among patients with STEMI was 6.2%. This mortality rate is comparable to that published in high-income and LMICs. An increase in heart rate and the administration of inotropes independently predicted all-cause mortality. At the same time, thrombolytic therapy and

percutaneous coro-nary intervention of the right coronary artery reduced mortality risk. In SSA, large prospective multicentre observational registries are still required to determine the mortality rate and define STEMI mortality predictors in our region.

REFERENCES

References

References

1. Belle L, Cayla G, Cottin Y, Coste P, Khalife K, Labeque JN, et al. French Registry on Acute ST-elevation and non-ST-elevation Myocardial Infarction 2015 (FAST-MI 2015). Design and baseline data. Arch Cardiovasc Dis. 2017;110(6-7):366-78.

2. Yao H, Ekou A, Niamkey T, Hounhoui Gan S, Kouame I, Afassinou Y, et al. Acute Coronary Syndromes in Sub-Saharan Africa: A 10-Year Systematic Review. J Am Heart Assoc. 2022;11(1):e021107.

3. Mensah GA. Descriptive epidemiology of cardiovascular risk factors and diabetes in sub-Saharan Africa. Prog Cardiovasc Dis. 2013;56(3):240-50.

4. Schamroth C, investigators ASA. Management of acute coronary syndrome in South Africa: insights from the ACCESS (Acute Coronary Events - a Multinational Survey of Current Management Strategies) registry. Cardiovasc J Afr. 2012;23(7):365-70.

5. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, et al. Third universal definition of myocardial infarction. Glob Heart. 2012;7(4):275-95.

6. Hertz JT, Reardon JM, Rodrigues CG, de Andrade L, Limkakeng AT, Bloomfield GS, et al. Acute myocardial infarction in sub-Saharan Africa: the need for data. PLoS One. 2014;9(5):e96688.

7. Seedat YK, Mayet FG, Latiff GH, Joubert G. Risk factors and coronary heart disease in Durban blacks--the missing links. S Afr Med J. 1992;82(4):251-6.

8. Moran A, Forouzanfar M, Sampson U, Chugh S, Feigin V, Mensah G. The epidemiology of cardiovascular diseases in sub-Saharan Africa: the Global Burden of Diseases, Injuries and Risk Factors 2010 Study. Prog Cardiovasc Dis. 2013;56(3):234-9.

9. Mensah GA, Roth GA, Sampson UK, Moran AE, Feigin VL, Forouzanfar MH, et al. Mortality from cardiovascular diseases in sub-Saharan Africa, 1990-2013: a systematic analysis of data from the Global Burden of Disease Study 2013. Cardiovasc J Afr. 2015;26(2 Suppl 1):S6-10.

10. Nkoke C, Luchuo EB. Coronary heart disease in sub-Saharan Africa: still rare, misdiagnosed or underdiagnosed? Cardiovasc Diagn Ther. 2016;6(1):64-6.

11. Kakou-Guikahue M, N'Guetta R, Anzouan-Kacou JB, Kramoh E, N'Dori R, Ba SA, et al. Optimising the management of acute coronary syndromes in sub-Saharan Africa: A statement from the AFRICARDIO 2015 Consensus Team. Arch Cardiovasc Dis. 2016;109(6-7):376-83.

12. Atun R, Davies JI, Gale EAM, Barnighausen T, Beran D, Kengne AP, et al. Diabetes in sub-Saharan Africa: from clinical care to health policy. Lancet Diabetes Endocrinol. 2017;5(8):622-67.

13. Hendriks ME, Wit FW, Roos MT, Brewster LM, Akande TM, de Beer IH, et al. Hypertension in sub-Saharan Africa: cross-sectional surveys in four rural and urban communities. PLoS One. 2012;7(3):e32638.

14. Noubiap JJ, Bigna JJ, Nansseu JR, Nyaga UF, Balti EV, Echouffo-Tcheugui JB, et al. Prevalence of dyslipidaemia among adults in Africa: a systematic review and meta-analysis. Lancet Glob Health. 2018;6(9):e998-e1007.

15. Guthold R, Louazani SA, Riley LM, Cowan MJ, Bovet P, Damasceno A, et al. Physical activity in 22 African countries: results from the World Health Organization STEPwise approach to chronic disease risk factor surveillance. Am J Prev Med. 2011;41(1):52-60.

16. GBD 2015 Obesity Collaborators, Afshin A, Forouzanfar MH, et al. Health Effects of Overweight and Obesity in 195 Countries over 25 Years. N Engl J Med. 2017;377(1):13-27.

17. Ebireri J, Aderemi AV, Omoregbe N, Adeloye D. Interventions addressing risk factors of ischaemic heart disease in sub-Saharan Africa: a systematic review. BMJ Open. 2016;6(7):e011881.

18. Wekesah FM, Kyobutungi C, Grobbee DE, Klipstein-Grobusch K. Understanding of and perceptions towards cardiovascular diseases and their risk factors: a qualitative study among residents of urban informal settings in Nairobi. BMJ Open. 2019;9(6):e026852.

19. Mohanan PP, Mathew R, Harikrishnan S, Krishnan MN, Zachariah G, Joseph J, et al. Presentation, management, and outcomes of 25 748 acute coronary syndrome admissions in Kerala, India: results from the Kerala ACS Registry. Eur Heart J. 2013;34(2):121-9.

20. Li J, Li X, Wang Q, Hu S, Wang Y, Masoudi FA, et al. ST-segment elevation myocardial infarction in China from 2001 to 2011 (the China PEACE-Retrospective Acute Myocardial Infarction Study): a retrospective analysis of hospital data. Lancet. 2015;385(9966):441-51.

21. Ali M, Lange SA, Wittlinger T, Lehnert G, Rigopoulos AG, Noutsias M. In-hospital mortality after acute STEMI in patients undergoing primary PCI. Herz. 2018;43(8):741-5.

22. N'Guetta R, Ekou A, Yao H, Anzouan-Kacou JB, Gerardin B, Pilliere R, et al. [Percutaneous coronary intervention in the management of acute coronary syndromes in Ivory Coast: Challenges and outcomes]. Ann Cardiol Angeiol (Paris). 2018;67(4):244-9.

23. Stassen W, Wallis L, Lambert C, Castren M, Kurland L. Percutaneous coronary intervention still not accessible for many South Africans. Afr J Emerg Med. 2017;7(3):105-7.

24. Varwani MH, Jeilan M, Ngunga M, Barasa A. Outcomes in patients with acute coronary syndrome in a referral hospital in sub-Saharan Africa. Cardiovasc J Afr. 2019;30(1):29-33.

25. Nyaaba GN, Stronks K, de-Graft Aikins A, Kengne AP, Agyemang C. Tracing Africa's progress towards implementing the Non-Communicable Diseases Global action plan 2013-2020: a synthesis of WHO country profile reports. BMC Public Health. 2017;17(1):297.

26. Anand SS, Islam S, Rosengren A, Franzosi MG, Steyn K, Yusufali AH, et al. Risk factors for myocardial infarction in women and men: insights from the INTERHEART study. Eur Heart J. 2008;29(7):932-40.

27. Larsson B, Bengtsson C, Bjorntorp P, Lapidus L, Sjostrom L, Svardsudd K, et al. Is abdominal body fat distribution a major explanation for the sex difference in the incidence of myocardial infarction? The study of men born in 1913 and the study of women, Goteborg, Sweden. Am J Epidemiol. 1992;135(3):266-73.

28. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937-52.

29. Helgestad OKL, Josiassen J, Hassager C, Jensen LO, Holmvang L, Sorensen A, et al. Temporal trends in incidence and patient characteristics in cardiogenic shock following acute myocardial infarction from 2010 to 2017: a Danish cohort study. Eur J Heart Fail. 2019;21(11):1370-8.

30. Granger CB, Goldberg RJ, Dabbous O, Pieper KS, Eagle KA, Cannon CP, et al. Predictors of hospital mortality in the global registry of acute coronary events. Arch Intern Med. 2003;163(19):2345-53.

31. Jensen MT, Pereira M, Araujo C, Malmivaara A, Ferrieres J, Degano IR, et al. Heart rate at admission is a predictor of in-hospital mortality in patients with acute coronary syndromes: Results from 58 European hospitals: The European Hospital

Benchmarking by Outcomes in acute coronary syndrome Processes study. Eur Heart J Acute Cardiovasc Care. 2018;7(2):149-57.

32. Morrow DA, Cannon CP, Jesse RL, Newby LK, Ravkilde J, Storrow AB, et al. National Academy of Clinical Biochemistry Laboratory Medicine Practice Guidelines: Clinical characteristics and utilisation of biochemical markers in acute coronary syndromes. Circulation. 2007;115(13):e356-75.

33. Khullar N, Buckley AJ, O'Connor C, Ibrahim A, Ibrahim A, Ahern C, et al. Peak troponin T in STEMI: a predictor of all-cause mortality and left ventricular function. Open Heart. 2022;9(1).

34. Wanamaker BL, Seth MM, Sukul D, Dixon SR, Bhatt DL, Madder RD, et al. Relationship Between Troponin on Presentation and In-Hospital Mortality in Patients With ST-Segment-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. J Am Heart Assoc. 2019;8(19):e013551.

35. Shouval R, Hadanny A, Shlomo N, Iakobishvili Z, Unger R, Zahger D, et al. Machine learning for prediction of 30-day mortality after ST elevation myocardial infraction: An Acute Coronary Syndrome Israeli Survey data mining study. Int J Cardiol. 2017;246:7-13.

36. Danchin N, Puymirat E, Steg PG, Goldstein P, Schiele F, Belle L, et al. Five-year survival in patients with ST-segment-elevation myocardial infarction according to modalities of reperfusion therapy: the French Registry on Acute ST-Elevation and Non-ST-Elevation Myocardial Infarction (FAST-MI) 2005 Cohort. Circulation. 2014;129(16):1629-36.

37. Meel R, Goncalves R. Time to fibrinolytics for acute myocardial infarction: Reasons for delays at Steve Biko Academic Hospital, Pretoria, South Africa. S Afr Med J. 2015;106(1):92-6.

Footnotes

Institutional review board statement: This study was reviewed and approved by the Ethics Committee of the University of Witwatersrand. Clearance certificate number: M201095

Informed consent statement: Patients were not required to give informed consent to the study as the study was a retrospective chart review

Conflict-of-interest statement: Professor Tsabedze is a consultant cardiologist. He has received consultation fees from Novartis Pharmaceuticals, Novo Nordisk, Boston Scientific, Pfizer, Servier, Phillips, Takeda, AstraZeneca, Acino Health Care Group and Merck. He has also received educational and travel grants from Medtronic, Biotronik, Boston Scientific and Vertice Health Care Group.

Data sharing statement: No additional data are available.

Supplementary table:

Lead territories

Variable	Frequency (%)
Lead territory	
Anterior	274 (40.47)
Anterior inferior	10 (1.48)
Anterior lateral	60 (8.86)
Anterior lateral inferior	6 (0.89)
Anterior posterior lateral	1 (0.15)
Inferior	232 (34.27)
Inferior lateral	36 (5.32)
Inferior lateral posterior	5 (0.74)
Inferior posterior	16 (2.36)
Lateral	15 (2.22)
None	19 (2.81)
Posterior	2 (0.30)
Posterior lateral	1 (0.15)

IN-HOSPITAL MORTALITY OUTCOMES FOR ACUTE ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

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ABBREVIATIONS AND ACRONYMS

ACS - Acute Coronary Syndrome

CMJAH - Charlotte Maxeke Johannesburg Academic Hospital

DAPT - dual antiplatelet therapy

ECG – Electrocardiogram

eGFR - estimated glomerular filtration rate

EMS - Emergency Medical System

FMC - first medical contact

Hb - Haemoglobin

IHD - Ischaemic Heart Disease

LBBB - left bundle branch block

MS - Metabolic Syndrome

MI - Myocardial infarction

NSTEMI - non-ST segment elevation myocardial infarction

PI - Principal Investigator

PCI - percutaneous coronary intervention

STEMI - ST-segment elevation myocardial infarction

SSA - Sub-Saharan Africa

1. INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) is a lethal and clinically identifiable consequence of ischaemic heart disease (IHD). STEMI mortality is on an upward trend in sub-Saharan Africa (SSA), which is in contrast to reports from western countries where STEMI mortality is decreasing(1)**.** There is a paucity of data published on the rising burden of this disease. Traditionally, the focus was on investigating the impact of communicable diseases, which still form a significant disease burden in SSA. Our study aims to determine the in-hospital mortality rate of STEMI at the Charlotte Maxeke Johannesburg Academic Hospital (CMJAH).

2. BACKGROUND AND LITERATURE REVIEW

2.1 ST-elevation myocardial infarction at a glance

Ischaemic heart disease has a vast, dynamic nature and can manifest as acute coronary syndromes (ACS). ST-segment elevation myocardial infarction is an ACS that displays clinical and biochemical evidence of myocardial necrosis (represented by raised cardiac troponin values with at least one value above the 99th percentile of the upper reference limit)(2). The acute nature of the event is represented by a rise and a fall in the cardiac troponin value. A distinct feature of STEMI is ST-segment elevation or new left bundle branch block (LBBB) on the electrocardiogram(3).

2.2 Diagnosis of an ST-elevation myocardial infarction

A diagnosis of STEMI requires symptoms consistent with STEMI, such as typical crushing chest pain and 12 lead ECG features. Some patients have atypical symptoms such as shortness of breath, nausea/vomiting, fatigue, palpitations, or syncope. Acute STEMI diagnosis is the time when the ECG is determined to have ST-segment elevation or equivalent, and it is the time “zero” to evidence-based guideline-mandated therapy.

2.3 ST-elevation myocardial infarction risk factors

The INTERHEART study, which reviewed risk factors for first myocardial infarction in 52 countries and enrolled over 27,000 subjects, concluded that nine factors were responsible for 90% of the population attributable risk (PAR) in all the specified regions. These factors were six harmful risk factors (dyslipidaemia, smoking, hypertension, diabetes mellitus, abdominal obesity, and stressful psychosocial factors) and three protective factors (daily consumption of fruits and vegetables, regular alcohol consumption in moderation, and regular physical activity)(4).

2.4 Epidemiology of ST-elevation myocardial infarction

There has been a significant reduction in the mortality rate of STEMI in developed countries(5). Data from the French Registry of Acute ST-segment elevation and Non-ST- segment elevation Myocardial Infarction (FAST-MI) of 2015 reported that STEMI mortality rate had significantly dropped, from 10.2% in 1995 to 2.1% in 2015(5). Contrary to this trend, a few studies in SSA have reported a high in-hospital mortality rate in patients with STEMI(6). Bahiru et al. conducted a retrospective review of ACS cases managed at Kenyatta National Hospital between 2013 and 2016. This study revealed that among 196 ACS admissions, the in-hospital mortality rate of individuals presenting with STEMI was 21%(7).

Acute STEMI incidence increases with age, and the risk of mortality increases proportionately(8). When compared to men with STEMI, women with STEMI have a higher mortality rate(9) due to women generally being older and having more co-morbidities at the time of STEMI diagnosis.

2.5 Clinical determinants of mortality

Cardiogenic shock is the chief cause of death in patients with acute STEMI. It complicates 5-8% of STEMIs, coupled with a mortality rate approaching 50%(10).

Mortality is significantly higher among patients with diabetes when compared to non-diabetics(11).

Patients presenting with renal dysfunction or anaemia also have an increased mortality rate. Renal dysfunction is a common co-morbidity and affects 30%-40% of patients with myocardial infarction, resulting in a worse prognosis(12)**.** Similarly, anaemia is a significant contributor to STEMI mortality. A baseline haemoglobin value <10 g/dL is associated with an approximately ten times higher mortality rate(13).

Cardiac arrest is also a common outcome in acute STEMI, generally resulting from ventricular fibrillation(14). Ventricular tachyarrhythmias are often an early consequence of STEMI and are also associated with a significantly increased risk of mortality(15).

Several traditional risk factors for STEMI improve in-hospital mortality outcomes. Recent data suggest that high blood pressure may be cardioprotective in the acute STEMI setting. A systolic blood pressure (SBP) <105 mmHg and a heart rate of ≥80 beats per minute on the admission of STEMI patients are associated with a higher risk of in-hospital mortality, even after primary PCI(16).

Two remarkable phenomena known as the obesity paradox and the hyperlipidaemia paradox exist in the literature. Here, there is an inverse association where hyperlipidaemia and obesity counterintuitively conferred an overall survival benefit in patients with STEMI(17, 18).

2.6 Pre-hospital delays and st-elevation myocardial infarction mortality

A study by Żurowska-Wolak et al. concluded that an increased length of time to first medical contact resulted in reduced ejection fraction and increased in-hospital mortality(19). The results of this study emphasise the importance of reducing delays to reperfusion therapy in STEMI patients. The recommended mode of patient presentation, which shortens delays to treatment, is by alerting the emergency medical service (EMS)(20). Diagnosis of STEMI in the pre-hospital setting should also prompt immediate activation of the catheterisation laboratory as this also reduces treatment delays and, subsequently, patient mortality(21).

2.7 Management and st-elevation myocardial infarction mortality

Early percutaneous coronary intervention (PCI) results in reduced mortality(22). It is the reperfusion strategy of choice in patients with STEMI, provided it can be performed expeditiously (i.e., 120 min from STEMI diagnosis). In specific scenarios, treating with fibrinolytic is appropriate where PCI is not immediately available. Fibrinolysis is a vital reperfusion strategy that prevents early mortality in 30 per 1000 patients treated within 6 hours after symptom onset(23). It is the current standard of care in many economically developing regions where there are significant pre-hospital delays and limited availability of fully equipped cardiac catheterisation units able to offer a 24 hour primary PCI service.

Several studies have supported radial access as the default access route in STEMI. In the Minimising Adverse Haemorrhagic Events by TRansradial Access Site and Systemic Implementation of angioX (MATRIX) study, radial access was associated with a significant morbidity and mortality benefit(24). Furthermore, the enhanced use of primary and secondary prevention therapy (dual antiplatelet therapy, statins, beta-blockers, and renin-angiotensin-aldosterone system inhibition) decreases mortality(25). All patients should receive dual antiplatelet therapy (DAPT). Combining aspirin and a P2Y12 inhibitor such as prasugrel, ticagrelor, or clopidogrel, is recommended in patients with STEMI. The omission of clopidogrel is a significant predictor of mortality(26). The use of high-intensity statin therapy, angiotensin-converting enzyme inhibitors, and beta-blockers are also associated with a significant mortality benefit and should be initiated early in patients without contraindications.

3. AIMS AND OBJECTIVES

3.1 Study aim

The majority of knowledge on IHD and STEMI is primarily from economically developed countries. This retrospective study aims to describe the clinical profiles and in-hospital mortality outcomes for STEMI patients treated in the division of cardiology at the CMJAH.

3.2 Study objectives

Primary:

- To determine the STEMI in-hospital mortality rate at CMJAH

Secondary:

- To describe the demographics of patients with STEMI in CMJAH

- To determine the average time duration (days) from symptom onset to reperfusion therapy and coronary angiography with or without PCI.
- To describe adjunct treatment received during the management of STEMI and on hospital discharge.
- To determine the predictors of in-hospital mortality in patients with STEMI at CMJAH

4. METHODOLOGY

4.1 Study design

Retrospective single centre review of all confirmed STEMIs at CMJAH between January 2015 – December 2019.

4.2 Study setting

Tertiary Center, public health facility, Division of Cardiology, Charlotte Maxeke Academic Hospital, Johannesburg, Gauteng.

4.3 Study population and sample size

A minimum, of 200 study participants with a confirmed STEMI diagnosis, from CMJAH, will be included.

Inclusion criteria

- Age >18
- Confirmed STEMI (Universal definition of MI)

Exclusion criteria

- Incomplete medical records
- Trauma-related myocardial infarction

4.4 Data collection

Relevant data from participants meeting the inclusion criteria above will be obtained from the electronic records of the cardiology department at CMJAH by the principal investigator (PI). The PI will create a reference list containing the participant's hospital number and an assigned study number. Once entered into the study dataset, participants will only be identifiable with the assigned study number. The reference list will only be available to the principal investigator of this study to maintain patient confidentiality. All patient identifiers will be removed once project moves to data analysis. The PI will use the Research Electronic Data Capture

(REDCap) tool as the data collection software. Research data will be collected from the 1st of January 2015 to the 31st of December 2019. The study data collection sheet (see appendix A) lists all the study variables that will be collected from the cardiology medical records.

5. DATA ANALYSIS

For this retrospective study, we will use descriptive statistics-frequency tables to describe clinical and demographic parameters. Means and their standard deviations will be used to summarise normally distributed continuous data. Medians and interquartile (IQR) ranges will be used to present continuous skewed data. Numbers and percentages will be used to summarise categorical data. The data will compare two groups consisting of those who survive in-hospital and those who demise. The Student t-test will be used for comparing means from normally distributed continuous variables, and the Wilcoxon rank-sum test will be used for comparing medians from non-normal continuous data. Multivariate logistic regression analyses to determine the predictors of in-hospital mortality will be done. These variables will be presented with their odds ratios and the 95% confidence interval. Statistical significance will be considered at a p-value < 0.05. Stata Corp. MP Version 16 will be used for computing the statistical data.

6. ETHICS

This study will be conducted at the Charlotte Maxeke Johannesburg Academic Hospital in the Department of Cardiology in fulfillment 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. 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

7. TIMING

	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEPT	OCT	DEC	JAN	FEB	MAR
Literature review													
Preparing protocol													
Protocol assessment													
Ethics Application													
Collecting data													
Data analysis													
Writing up – thesis													
Writing up – paper													

8. FUNDING

The research project is a low-cost endeavor. The PI will be able to meet the running expenses, which include stationery, printing, food, and transport.

9. ANTICIPATED LIMITATIONS

- Lack of prior research studies on the topic: extensive literature review revealed minimal data
- Record keeping: incomplete echo, admission records

REFERENCES:

1. Cappuccio FP, Miller MA. Cardiovascular disease and hypertension in sub-Saharan Africa: burden, risk and interventions. Intern Emerg Med. 2016;11(3):299-305.

2. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction (2018). Circulation. 2018;138(20):e618-e51.

3. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Rev Esp Cardiol (Engl Ed). 2017;70(12):1082.

4. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937-52.

5. Belle L, Cayla G, Cottin Y, Coste P, Khalife K, Labeque JN, et al. French Registry on Acute ST-elevation and non-ST-elevation Myocardial Infarction 2015 (FAST-MI 2015). Design and baseline data. Arch Cardiovasc Dis. 2017;110(6-7):366-78.

6. N'Guetta R, Yao H, Brou I, Ekou A, Do P, Angoran I, et al. [Prevalence and characteristics of metabolic syndrome among hypertensive patients in Abidjan]. Ann Cardiol Angeiol (Paris). 2016;65(3):131-5.

7. Bahiru E, Temu T, Gitura B, Farquhar C, Huffman MD, Bukachi F. Presentation, management and outcomes of acute coronary syndrome: a registry study from Kenyatta National Hospital in Nairobi, Kenya. Cardiovasc J Afr. 2018;29(4):225-30.

8. Goldberg RJ, McCormick D, Gurwitz JH, Yarzebski J, Lessard D, Gore JM. Age-related trends in short- and long-term survival after acute myocardial infarction: a 20-year population-based perspective (1975-1995). Am J Cardiol. 1998;82(11):1311-7.

9. Berger JS, Elliott L, Gallup D, Roe M, Granger CB, Armstrong PW, et al. Sex differences in mortality following acute coronary syndromes. JAMA. 2009;302(8):874-82.

10. Babaev A, Frederick PD, Pasta DJ, Every N, Sichrovsky T, Hochman JS, et al. Trends in management and outcomes of patients with acute myocardial infarction complicated by cardiogenic shock. JAMA. 2005;294(4):448-54.

11. Donahoe SM, Stewart GC, McCabe CH, Mohanavelu S, Murphy SA, Cannon CP, et al. Diabetes and mortality following acute coronary syndromes. JAMA. 2007;298(7):765-75.

12. Szummer K, Lundman P, Jacobson SH, Schon S, Lindback J, Stenestrand U, et al. Relation between renal function, presentation, use of therapies and in-hospital complications in acute coronary syndrome: data from the SWEDEHEART register. J Intern Med. 2010;268(1):40-9.

13. Wester A, Attar R, Mohammad MA, Andell P, Hofmann R, Jensen J, et al. Impact of Baseline Anemia in Patients With Acute Coronary Syndromes Undergoing Percutaneous Coronary Intervention: A Prespecified Analysis From the VALIDATE-SWEDEHEART Trial. J Am Heart Assoc. 2019;8(16):e012741.

14. Larsen JM, Ravkilde J. Acute coronary angiography in patients resuscitated from out-of-hospital cardiac arrest--a systematic review and meta-analysis. Resuscitation. 2012;83(12):1427-33.

15. Henkel DM, Witt BJ, Gersh BJ, Jacobsen SJ, Weston SA, Meverden RA, et al. Ventricular arrhythmias after acute myocardial infarction: a 20-year community study. Am Heart J. 2006;151(4):806-12.

16. Bordejevic DA, Caruntu F, Mornos C, Olariu I, Petrescu L, Tomescu MC, et al. Prognostic impact of blood pressure and heart rate at admission on in-hospital mortality after primary percutaneous intervention for acute myocardial infarction with ST-segment elevation in western Romania. Ther Clin Risk Manag. 2017;13:1061-8.

17. Angeras O, Albertsson P, Karason K, Ramunddal T, Matejka G, James S, et al. Evidence for obesity paradox in patients with acute coronary syndromes: a report from the Swedish Coronary Angiography and Angioplasty Registry. Eur Heart J. 2013;34(5):345-53.

18. Yousufuddin M, Takahashi PY, Major B, Ahmmad E, Al-Zubi H, Peters J, et al. Association between hyperlipidemia and mortality after incident acute myocardial infarction or acute decompensated heart failure: a propensity score matched cohort study and a meta-analysis. BMJ Open. 2019;9(12):e028638.

19. Zurowska-Wolak M, Piekos P, Jakala J, Mikos M. The effects of prehospital system delays on the treatment efficacy of STEMI patients. Scand J Trauma Resusc Emerg Med. 2019;27(1):39.

20. Perkins-Porras L, Whitehead DL, Strike PC, Steptoe A. Pre-hospital delay in patients with acute coronary syndrome: factors associated with patient decision time and home-to-hospital delay. Eur J Cardiovasc Nurs. 2009;8(1):26-33.

21. Stowens JC, Sonnad SS, Rosenbaum RA. Using EMS Dispatch to Trigger STEMI Alerts Decreases Door-to-Balloon Times. West J Emerg Med. 2015;16(3):472-80.

22. Huynh T, Perron S, O'Loughlin J, Joseph L, Labrecque M, Tu JV, et al. Comparison of primary percutaneous coronary intervention and fibrinolytic therapy in ST-segment-elevation myocardial infarction: bayesian hierarchical meta-analyses of randomised controlled trials and observational studies. Circulation. 2009;119(24):3101-9.

23. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Lancet. 1994;343(8893):311-22.

24. Valgimigli M, Gargiulo G, Juni P. Radial versus femoral access for cardiac catheterisation - Authors' reply. Lancet. 2015;386(10011):2394.

25. Szummer K, Jernberg T, Wallentin L. From Early Pharmacology to Recent Pharmacology Interventions in Acute Coronary Syndromes: JACC State-of-the-Art Review. J Am Coll Cardiol. 2019;74(12):1618-36.

26. Chen ZM, Jiang LX, Chen YP, Xie JX, Pan HC, Peto R, et al. Addition of clopidogrel to aspirin in 45,852 patients with acute myocardial infarction: randomised placebo-controlled trial. Lancet. 2005;366(9497):1607-21.

Patient Details

Record ID	_____
Patient name	_____
Patient surname	_____
Hospital number	_____

Demographics

Date of admission	_____
Sex	◯ Male ◯ Female
Date of birth	_____
Age at time of admission to hospital	_____
Race	◯ African ◯ White ◯ Asian ◯ Coloured ◯ Other
Family history of heart disease	◯ Yes ◯ No
Co-morbidities	☐ hypertension ☐ diabetes mellitus ☐ dyslipidaemia ☐ obesity ☐ chronic kidney disease ☐ peripheral vascular disease ☐ Coronary artery disease ☐ Human immunodeficiency virus (HIV) ☐ other (Based on known disease and disease confirmed on admission. Obesity = BMI of 30 or more and clinicians clinical judgement. Dydlipidaemia=increased low-density lipoprotein cholesterol, decreased high-density lipoprotein cholesterol, and increased serum triglycerides)
Level of activity	◯ Little or no exercise ◯ Exercise 1-3 times/week ◯ Exercise 4-5 times/week ◯ Daily exercise or intense exercise 3-4 times/week ◯ Intense exercise 6-7 times/week ◯ very intense exercise daily, or physical job ◯ Not documented (Exercise: 15-30 minutes of elevated heart rate activity. Intense exercise: 45-120 minutes of elevated heart rate activity. Very intense exercise: 2+ hours of elevated heart rate activity.)
Psychosocial stressors	◯ Yes ◯ No ◯ Not documented

Mode of Presentation

Typical chest pain	◯ Yes ◯ No
Date and time of onset of chest pain	_____
Time to first medical contact(days)	_____
Duration between onset of chest pain and first medical contact	_____
Date and time of angiogram	_____
Duration between onset of chest pain and cardiac catheterization	_____

ECG Changes

Sinus rhythm	◯ Yes ◯ No
Abnormal rhythm	☐ Atrial fibrillation ☐ Atrial flutter ☐ Ventricular tachycardia ☐ Ventricular fibrillation ☐ Heart block ☐ Cardiac arrest ☐ Sinus bradycardia ☐ Premature beats ☐ Paroxysmal tachycardia
Heart Rate (bpm)	_____
ST elevation	☐ Anterior leads ☐ Lateral leads ☐ Inferior leads ☐ Posterior
Q waves	◯ Yes ◯ No
Location of Q waves	Anterior Inferior Posterior Lateral

Clinical Parameters

Systolic blood pressure at presentation	_____
Diastolic blood pressure at presentation	_____
NYHA classification at presentation	◯ Class I ◯ Class II ◯ Class III ◯ Class IV (I No limitation of physical activity. II Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea. III Marked limitation of physical activity. Comfortable at rest. Less than ordinary activity causes fatigue, palpitation, or dyspnea. IV Unable to carry on any physical activity without discomfort. Symptoms of heart failure at rest. If any physical activity is undertaken, discomfort increases.)
Killip class	◯ No signs of congestion ◯ S3 and basal rales on auscultation ◯ Acute pulmonary edema ◯ Cardiogenic shock

Biochemical Parameters

Peak troponin T	_____
Peak Trop I	_____
CK-MB	_____
Haemoglobin (HB)	_____
Sodium	_____
Potassium	_____
Urea	_____
Creatinine	_____
eGFR (MDRD equation)	_____
Total cholesterol	_____
Triglycerides	_____
LDL	_____
HDL	

Angiography Parameters

Dominance	◯ RIght ◯ Left ◯ Co-dominance
Vessel(s) involvement	☐ Right coronary artery ☐ Left main coronary artery ☐ Left circumflex artery ☐ Left anterior descending artery ☐ Posterior descending artery ☐ OM/Ramus ☐ D1 ☐ No lesion

Intervention

Adjuncts Recieved	☐ Aspirin ☐ Clopidogrel ☐ Clexane ☐ Statin
Thrombolysed	◯ Yes ◯ No
Date and time thrombolysed	_____
Inotropes	◯ Yes ◯ No
Inotrope received	Dobutamine Adrenaline ☐ Phenylephrine ☐ ☐
Pacing	◯ Yes ◯ No
Percutaneous coronary intervention	◯ Yes ◯ No
Percutaneous coronary intervention performed on:	☐ Right coronary artery ☐ Left main coronary artery ☐ Left circumflex artery ☐ Left anterior descending artery ☐ Posterior descending artery ☐ Obtuse marginal artery ☐ OM Ramus

Post-intervention Complications

Arrhythmia	◯ Yes ◯ No
Specify arrhythmia	☐ Atrial fibrillation ☐ Ventricular fibrillation ☐ Atrial flutter ☐ Ventricular tachycardia ☐ Premature ventricular contractions ☐ Asystole ☐ Heart block ☐ Other

Discharge Medication

Dual antiplatelet therapy	◯ No ◯ Yes
Dual antiplatelet therapy total daily dose	_____
Aspirin	◯ No ◯ Yes
Aspirin total daily dose	_____
Clopidogrel	◯ No ◯ Yes
Clopidogrel total daily dose	_____
Beta blocker	◯ No ◯ Yes
Beta blocker total daily dose	_____
Statin	◯ No ◯ Yes
Statin total daily dose	_____
ACE inhibitor	◯ No ◯ Yes
ACE-I total daily dose	_____
Angiotensin receptor blocker antagonists	No ◯ Yes ◯
Angiotensin receptor blocker total daily dose	
Mineralocorticoid receptor antagonist	No ◯ Yes ◯
Mineralocorticoid receptor antagonist total daily dose	

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

FACULTY OF HEALTH SCIENCES

University of the Witwatersrand Student Ethics Declaration Form

(To be completed during the protocol assessor meeting)

Background

All Research conducted by a University of the Witwatersrand student, with human subjects or animals, requires approval by the Wits Human Research Ethics Committee or Animal Research Ethics Committee, respectively.

If research has been undertaken without the necessary ethics approvals, this is considered an ethics violation. This will be reported to the relevant structures, the data will have to be discarded, and in the case of students, they cannot use the data towards their degree.

To prevent any ethics violations, the ethics requirements for the proposed project will be discussed with you at the protocol assessment.

Declaration

Based on the current protocol assessment (and any proposed changes suggested by the assessor committee), we, the undersigned, understand that the proposed research requires:

1. Human Research Ethics clearance certificate — Yes / No
    a. Covered under existing supervisor ethics — Yes / No
    b. Requires a new HREC application — Yes / No
2. Animal Research Ethics clearance certificate — Yes / No
3. No Human or Animal Ethics Clearance — Yes / No
4. Unclear, will seek appropriate guidance from the HREC/AREC committees (whichever relevant) — Yes / No

Signatures

Supervisor/s: Arthur Mutyaba

Student: L.W. Ndaba

Date: 03 Sept 2020

11 March 2019/MP

UNIVERSITY OF THE
WITWATERSRAND
JOHANNESBURG

R14/49 Dr L Ndaba

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
CLEARANCE CERTIFICATE NO. M201095

NAME: (Principal Investigator)	Dr L Ndaba
DEPARTMENT:	School of Clinical Medicine Department of Medicine Division of Internal Medicine Medical School University
PROJECT TITLE:	In-hospital mortality outcomes for acute St-Segment elevation myocardial infarction
DATE CONSIDERED:	30/10/2020
DECISION:	Approved unconditionally
CONDITIONS:	
SUPERVISOR:	Drs N Tsabedze and A Mutyaba
APPROVED BY:	Dr CB Penny, Chairperson, HREC (Medical)
DATE OF APPROVAL:	19 November 2020

This clearance certificate is valid for 5 years from the date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and **ONE COPY** returned to the Research Office Secretary on the 3rd Floor, Phillip Tobias Building, Parktown, University of the Witwatersrand, Johannesburg.
I/we fully understand the conditions under which I am/we are authorized to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated, from the research protocol as approved, I/we undertake to submit details to the Committee. **I agree to submit a yearly progress report**. When a funder requires annual re-certification, the application date will be one year after the date when the study was initially reviewed. In this case, the study was initially reviewed in **October** and will therefore reports and re-certification will be due early in the month of **October** each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).

Principal Investigator Signature

24/11/2020
Date

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by Lindokuhle Ndaba

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Column: Retrospective Study

Authors: Lindokuhle Wiseman Ndaba, Dineo Mpanya, Arthur Mutyaba and Nqoba Tsabedze

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