

https://dspace.org/

The Prevalence of Arrhythmias in Acute ST-Segment Elevation Myocardial Infarction

Setshego, Mothusi Koketso

2024
University of the Witwatersrand, Johannesburg
https://hdl.handle.net/10539/42420
Setshego, Mothusi Koketso. (2024). The role of design houses [Master's dissertation , University of the Witwatersrand, Johannesburg]. WireDSpace.

Downloaded from a DSpace repository. https://dspace.org/

COVER PAGE

The Prevalence of Arrhythmias in Acute ST-Segment Elevation Myocardial Infarction

MOTHUSI KOKETSO SETSHEGO, Bsc (UB), MBCHB(UKZN), FCP(SA)

Student number:2417975

Supervisors:

Professor Nqoba Tsabedze, MBBCh (Wits), MMed (Wits) ,FCP (SA), Cert Cardiology (SA)

Dr Anupa Patel, MBBCh (Wits), FCP(SA), Cert Cardiology(SA)

A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand,

Johannesburg, in partial fulfilment of the requirements for the degree of Master of Medicine(Internal Medicine)Johannesburg

29 JANUARY 2024

Declaration

I, Dr Mothusi Koketso Setshego, declare that this research report (submissible article and protocol with comprehensive literature review) is my unaided work. It is being submitted for the Degree of Master of Medicine at the University of the Witwatersrand. It has not been previously submitted for any other degree or examination at this or any other University.

Signature of the student

_____ Date: 22/01/2024

ACKNOWLEDGMENTS

To my family and friends, thank you for all your support. To my supervisors, I will forever be eternally grateful for your all the time and effort you have shown me throughout the years in writing this paper.

To my wife Nabeelah Allie-Setshego, thank you for being my pillar of strength.

Table of Contents

COVER PAGE....1
Declaration....2
CO-AUTHORS' AGREEMENT FOR WRITE-UP WITH SUBMISSIBLE ARTICLES....2
ACKNOWLEDGMENTS....3
ABSTRACT....5
RESEARCH REPORT....15
REFERENCES....26
APPENDICES....28
PROTOCOL....35

ABSTRACT

South Africa has a rising prevalence of ischaemic heart diseases. Patients with acute myocardial infarction who succumb to sudden cardiac death tend to have fatal arrhythmias. This study aims to determine the prevalence of arrhythmias in patients with acute ST-segment elevation myocardial infarction (STEMI). We retrospectively reviewed medical records of patients with STEMI at a tertiary academic hospital for a 5 year period between 1 January 2015 and 31 December 2019. The study population comprised 259 patients with a mean age of 56.0 ±10.8 years. Dyslipidaemia was the most common co-morbidity, found in 169 (65.2%) patients. There were 125 (48.3%), 123 (47.5%) and 11 (4.2%) patients with inferior, antero-septal and lateral myocardial infarction, respectively. The most common arrhythmia was a sinus tachycardia, found in 80 (12.1%) patients, followed by a sinus bradycardia in 37 (5.6%). Thirty-seven of the 80 (70.1%) patients with a sinus tachycardia had ST-elevation in the anterior leads, and 19(51.3%) with a sinus bradycardia had ST-elevation in the inferior leads ($p<0.009$). Among the 143 patients with left anterior descending artery stenosis, 72 (50.3%) had a sinus tachycardia, while 19 (13.3%) had a sinus bradycardia ($p<0.001$). The most common arrhythmias in STEMI patients were a sinus tachycardia and bradycardia, accounting for arrhythmias in 36 and 27% of all patients, respectively.

Conclusion:The most common arrhythmias in ST-elevation MI patients were a sinus tachycardia and bradycardia, accounting for arrhythmias in 36% and 27% of all patients, respectively. Future prospective and multicentre studies are required in low-and-middle-income countries, which will determine the true burden of arrhythmia in acute STEMI.

Keywords: myocardial infarction; electrocardiogram; arrhythmia

Author guidelines: Journal of Clinical Medicine

Manuscript Preparation

General Considerations

- **Research manuscripts** should comprise:

o **Front matter**: Title, Author list, Afiliations, Abstract, Keywords.

o **Research manuscript sections**: Introduction, Materials and Methods, Results, Discussion, Conclusions (optional).

o **Back matter**: Supplementary Materials, Acknowledgments, Author Contributions, Conflicts of Interest, **References**.

- **Review manuscripts** should comprise the **front matter**, literature review sections and the **back matter**. The template file can also be used to prepare the front and back matter of your review manuscript. It is not necessary to follow the remaining structure. Structured reviews and meta-analyses should use the same structure as research articles and ensure they conform to the **PRISMA** guidelines.

- **Graphical Abstract:**

A graphical abstract (GA) is an image that appears alongside the text abstract in the Table of Contents. In addition to summarising the content, it should represent the topic of the article in an attention-grabbing way. Moreover, it should not be exactly the same as the Figure in the paper or just a simple superposition of several subfigures. Note that the GA must be original and unpublished artwork. Any postage stamps, currency from any country, or trademarked items should not be included in it.

The GA should be a high-quality illustration or diagram in any of the following formats: PNG, JPEG, TIFF, or SVG. Written text in a GA should be clear and easy to read, using one of the following fonts: Times, Arial, Courier, Helvetica, Ubuntu or Calibri.

The minimum required size for the GA is 560 × 1100 pixels (height × width). The size should be of high quality in order to reproduce well.

- **Acronyms/Abbreviations/Initialisms** should be defined the first time they appear in each of three sections: the abstract; the main text; the first figure or table. When defined for the first time, the acronym/abbreviation/initialism should be added in parentheses after the written-out form.

- **SI Units** (International System of Units) should be used. Imperial, US customary and other units should be converted to SI units whenever possible.

- **Accession numbers** of RNA, DNA and protein sequences used in the manuscript should be provided in the Materials and Methods section. Also see the section on **Deposition of Sequences and Expression Data**.

- **Equations:** If you are using Word, please use either the Microsoft Equation Editor or the MathType add-on. Equations should be editable by the editorial ofice and not appear in a picture format.

Research Data and supplementary materials:

Note that publication of your manuscript implies that you must make all materials, data, and protocols associated with the publication available to readers. Disclose at the submission stage any restrictions on the availability of materials or information. Read the information about **Supplementary Materials** and Data Deposit for additional guidelines.

- **Preregistration:**

Where authors have preregistered studies or analysis plans, links to the preregistration must be provided in the manuscript.

- **Guidelines and standards:** MDPI follows standards and guidelines for certain types of research. See **https://www.mdpi.com/editorial_process** for further information.

Front Matter

These sections should appear in all manuscript types

- **Title:**

The title of your manuscript should be concise, specific and relevant. It should identify if the study reports (human or animal) trial data, or is a systematic review, meta-analysis or replication study. When gene or protein names are included, the abbreviated name rather than full name should be used. Please do not include abbreviated or short forms of the title, such as a running title or head. These will be removed by our Editorial Ofice.

- **Author List and Afiliations:**

Authors' full first and last names must be provided. The initials of any middle names can be added. The PubMed/MEDLINE standard format is used for afiliations: complete address information including city, zip code, state/province, and country. At least one author should be designated as the corresponding author. The email addresses of all authors will be displayed on published papers, and hidden by Captcha on the website as standard. It is the responsibility of the corresponding author to ensure that consent for the display of email addresses is obtained from all authors. If an author (other than the corresponding author) does not wish to have their email addresses displayed in this way, the corresponding author must indicate as such during proofreading. After acceptance, updates to author names or afiliations may not be permitted. Equal Contributions: authors who have contributed equally should be marked with a superscript symbol (†). The symbol must be included below the afiliations, and the following statement added: “These authors contributed equally to this work”. The equal roles of authors should also be adequately disclosed in the author contributions statement. Please read the criteria to qualify for authorship.

- **Keywords:**

Three to ten pertinent keywords need to be added after the abstract. We recommend that the keywords are specific to the article, yet reasonably common within the subject discipline.

Research Manuscript Sections

- **Introduction:**

The introduction should briefly place the study in a broad context and highlight why it is important. It should define the purpose of the work and its significance, including specific hypotheses being tested. The current state of the research field should be reviewed carefully and key publications cited. Please highlight controversial and diverging hypotheses when necessary. Finally, briefly mention the main aim of the work and highlight the main conclusions. Keep the introduction comprehensible to scientists working outside the topic of the paper.

- **Materials and Methods:**

They should be described with suficient detail to allow others to replicate and build on published results. New methods and protocols should be described in detail while well-established methods can be briefly described and appropriately cited. Give the name and version of any software used and make clear whether computer code used is available. Include any pre-registration codes.

- **Results:**

Provide a concise and precise description of the experimental results, their interpretation as well as the experimental conclusions that can be drawn.

- **Discussion:**

Authors should discuss the results and how they can be interpreted in perspective of previous studies and of the working hypotheses. The findings and their implications should be discussed in the broadest context possible and limitations of the work highlighted. Future research directions may also be mentioned. This section may be combined with Results.

- **Conclusions:** This section is not mandatory but can be added to the manuscript if the discussion is unusually long or complex.

Abstract: The abstract should be a total of about 200 words maximum. The abstract should be a single paragraph and should follow the style of structured abstracts, but without headings: 1) Background: Place the question addressed in a broad context and highlight the purpose of the study; 2) Methods: Describe briefly the main methods or treatments applied. Include any relevant preregistration numbers, and species and strains of any animals used; 3) Results: Summarise the article's main findings; and 4) Conclusion: Indicate the main conclusions or interpretations. The abstract should be an objective representation of the article: it must not contain results which are not presented and substantiated in the main text and should not exaggerate the main conclusions

- **Patents:** This section is not mandatory but may be added if there are patents resulting from the work reported in this manuscript

Back Matter

- **Supplementary Materials:** Describe any supplementary material published online alongside the manuscript (figure, tables, video, spreadsheets, etc.). Please indicate the name and title of each element as follows Figure S1: title, Table S1: title, etc.

- **Author Contributions:** Each author is expected to have made substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data; or the creation of new software used in the work; or have drafted the work or substantively revised it; AND has approved the submitted version (and version substantially edited by journal staff that involves the author's contribution to the study); AND agrees to be personally accountable for the author's own contributions and for ensuring that questions related to the accuracy or integrity of any part of the work, even ones in which the author was not personally involved, are appropriately investigated, resolved, and documented in the literature.
For research articles with several authors, a short paragraph specifying their individual

contributions must be provided. The following statements should be used "Conceptualization, X.X. and Y.Y.; Methodology, X.X.; Software, X.X.; Validation, X.X., Y.Y. and Z.Z.; Formal Analysis, X.X.; Investigation, X.X.; Resources, X.X.; Data Curation, X.X.; Writing – Original Draft Preparation, X.X.; Writing – Review & Editing, X.X.; Visualization, X.X.; Supervision, X.X.; Project Administration, X.X.; Funding Acquisition, Y.Y.", please turn to the **CRediT taxonomy** for the term explanation. For more background on CRediT, see **here.**

"**Authorship must include and be limited to those who have contributed substantially to the work. Please read the section concerning the criteria to qualify for authorship carefully**".

- **Funding:** All sources of funding of the study should be disclosed. Clearly indicate grants that you have received in support of your research work and if you received funds to cover publication costs. Note that some funders will not refund article processing charges (APC) if the funder and grant number are not clearly and correctly identified in the paper. Funding information can be entered separately into the submission system by the authors during submission of their manuscript. Such funding information, if available, will be deposited to FundRef if the manuscript is finally published.
Please add: “This research received no external funding” or “This research was funded by [name of funder] grant number [xxx]” and “The APC was funded by [XXX]” in this section. Check carefully that the details given are accurate and use the standard spelling of funding agency names at https://search.crossref.org/funding, any errors may affect your future funding.

- **Institutional Review Board Statement:** In this section, please add the Institutional Review Board Statement and approval number for studies involving humans or animals. Please note that the Editorial Ofice might ask you for further information. Please add “The study was conducted according to the guidelines of the Declaration of Helsinki, and approved by the Institutional Review Board (or Ethics Committee) of NAME OF INSTITUTE (protocol code XXX and date of approval).” OR “Ethical review and approval were waived for this study, due to REASON (please provide a detailed justification).” OR “Not applicable” for studies not involving humans or animals. You might also choose to exclude this statement if the study did not involve humans or animals.

- **Informed Consent Statement:** Any research article describing a study involving humans should contain this statement. Please add “Informed consent was obtained from all subjects involved in the study.” OR “Patient consent was waived due to REASON (please provide a detailed justification).” OR “Not applicable” for studies not involving humans. You might also choose to exclude this statement if the study did not involve humans.
Written informed consent for publication must be obtained from participating patients who can be identified (including by the patients themselves). Please state “Written informed consent has been obtained from the patient(s) to publish this paper” if applicable.

- **Data Availability Statement:** In this section, please provide details regarding where data supporting reported results can be found, including links to publicly archived datasets analysed or generated during the study. Please refer to suggested Data Availability Statements in section **“MDPI Research Data Policies”**. You might choose to exclude this statement if the study did not report any data.

Acknowledgments: In this section you can acknowledge any support given which is not covered by the author contribution or funding sections. This may include administrative and technical support, or donations in kind (e.g., materials used for experiments).

Conflicts of Interest: Authors must identify and declare any personal circumstances or interest that may be perceived as influencing the representation or interpretation of reported research results. If there is no conflict of interest, please state "The authors declare no conflict of interest." Any role of the funding sponsors in the choice of research project; design of the study; in the collection, analyses or interpretation of data; in the writing of the manuscript; or in the decision to publish the results must be declared in this section. *JCM* does not publish studies funded partially or fully by the tobacco industry. Any projects funded by industry must pay special attention to the full declaration of funder involvement. If there is no role, please state "The sponsors had no role in the design, execution, interpretation, or writing of the study". For more details please see **Conflict of Interest**.

References: References must be numbered in order of appearance in the text (including table captions and figure legends) and listed individually at the end of the manuscript. We recommend preparing the references with a bibliography software package, such as **EndNote**, **ReferenceManager** or **Zotero** to avoid typing mistakes and duplicated references. We encourage citations to data, computer code and other citable research material. If available online, you may use reference style 9. below.

Citations and References in Supplementary files are permitted provided that they also appear in the main text and in the reference list.

In the text, reference numbers should be placed in square brackets [ ], and placed before the punctuation; for example [1], [1–3] or [1,3]. For embedded citations in the text with pagination, use both parentheses and brackets to indicate the reference number and page numbers; for example [5] (p. 10). or [6] (pp. 101–105).

The reference list should include the full title, as recommended by the ACS style guide. Style files for **Endnote** and **Zotero** are available.

References should be described as follows, depending on the type of work:

Journal Articles:
1. Author 1, A.B.; Author 2, C.D. Title of the article. *Abbreviated Journal Name* **Year**, *Volume*, page range.

Books and Book Chapters:
2. Author 1, A.; Author 2, B. *Book Title*, 3rd ed.; Publisher: Publisher Location, Country, Year; pp. 154– 196.
3. Author 1, A.; Author 2, B. Title of the chapter. In *Book Title*, 2nd ed.; Editor 1, A., Editor 2, B., Eds.; Publisher: Publisher Location, Country, Year; Volume 3, pp. 154–196.

Unpublished materials intended for publication:
4. Author 1, A.B.; Author 2, C. Title of Unpublished Work (optional). Correspondence Afiliation, City, State, Country. year, *status* (*manuscript in preparation*; *to be submitted*).
5. Author 1, A.B.; Author 2, C. Title of Unpublished Work. *Abbreviated Journal Name* year, *phrase indicating stage of publication* (*submitted*; *accepted*; *in press*).

- **Acknowledgments:**

In this section you can acknowledge any support given which is not covered by the author contribution or funding sections. This may include administrative and technical support, or donations in kind (e.g., materials used for experiments).

- **Conflicts of Interest:**

Authors must identify and declare any personal circumstances or interest that may be perceived as influencing the representation or interpretation of reported research results. If there is no conflict of interest, please state "The authors declare no conflict of interest." Any role of the funding sponsors in the choice of research project; design of the study; in the collection, analyses or interpretation of data; in the writing of the manuscript; or in the decision to publish the results must be declared in this section. *JCM* does not publish studies funded partially or fully by the tobacco industry. Any projects funded by industry must pay special attention to the full declaration of funder involvement. If there is no role, please state "The sponsors had no role in the design, execution, interpretation, or writing of the study". For more details please see **Conflict of Interest**.

- **References:**

References must be numbered in order of appearance in the text (including table captions and figure legends) and listed individually at the end of the manuscript. We recommend preparing the references with a bibliography software package, such as **EndNote**, **ReferenceManager** or **Zotero** to avoid typing mistakes and duplicated references. We encourage citations to data, computer code and other citable research material. If available online, you may use reference style 9. below.

- Citations and References in Supplementary files are permitted provided that they also appear in the main text and in the reference list.

In the text, reference numbers should be placed in square brackets [ ], and placed before the punctuation; for example [1], [1–3] or [1,3]. For embedded citations in the text with pagination, use both parentheses and brackets to indicate the reference number and page numbers; for example [5] (p. 10). or [6] (pp. 101–105).

The reference list should include the full title, as recommended by the ACS style guide. Style files for **Endnote** and **Zotero** are available.

References should be described as follows, depending on the type of work:

 Journal Articles:
1. Author 1, A.B.; Author 2, C.D. Title of the article. *Abbreviated Journal Name* **Year**, *Volume*, page range.

 Books and Book Chapters:
2. Author 1, A.; Author 2, B. *Book Title*, 3rd ed.; Publisher: Publisher Location, Country, Year; pp. 154–196.
3. Author 1, A.; Author 2, B. Title of the chapter. In *Book Title*, 2nd ed.; Editor 1, A., Editor 2, B., Eds.; Publisher: Publisher Location, Country, Year; Volume 3, pp. 154–196.

4. Author 1, A.B.; Author 2, C. Title of Unpublished Work (optional). Correspondence Afiliation, City, State, Country. year, *status* (*manuscript in preparation*; *to be submitted*).

5. Author 1, A.B.; Author 2, C. Title of Unpublished Work. *Abbreviated Journal Name* year, *phrase indicating stage of publication* (*submitted*; *accepted*; *in press*).

6. Author 1, A.B.; Author 2, C. Title of Unpublished Work (optional). Correspondence Afiliation, City,State, Country. year, *status* (*manuscript in preparation*; *to be submitted*).

5. Author 1, A.B.; Author 2, C. Title of Unpublished Work. *Abbreviated Journal Name* year, *phrase indicating stage of publication* (*submitted*; *accepted*; *in press*).

7. Author 1, A.B.; Author 2, C. Title of Unpublished Work (optional). Correspondence Afiliation, City, State, Country. year, *status* (*manuscript in preparation*; *to be submitted*).
8. Author 1, A.B.; Author 2, C. Title of Unpublished Work. *Abbreviated Journal Name* year, *phrase indicating stage of publication* (*submitted*; *accepted*; *in press*).

9. Author 1, A.B.; Author 2, C. Title of Unpublished Work (optional). Correspondence Afiliation, City, State, Country. year, *status* (*manuscript in preparation*; *to be submitted*).
10. Author 1, A.B.; Author 2, C. Title of Unpublished Work. *Abbreviated Journal Name* year, *phrase indicating stage of publication* (*submitted*; *accepted*; *in press*).

11. Author 1, A.B.; Author 2, C. Title of Unpublished Work (optional). Correspondence Afiliation, City, State, Country. year, status (manuscript in preparation; to be submitted).
12. Author 1, A.B.; Author 2, C. Title of Unpublished Work. Abbreviated Journal Name year, phrase indicating stage of publication (submitted; accepted; in press).

13. Author 1, A.B.; Author 2, C. Title of Unpublished Work (optional). Correspondence Afiliation, City, State, Country. year, *status* (*manuscript in preparation*; *to be submitted*).

15. Author 1, A.B.; Author 2, C. Title of Unpublished Work. *Abbreviated Journal Name* year, *phrase indicating stage of publication* (*submitted*; *accepted*; *in press*).

 Unpublished materials not intended for publication:

16. Author 1, A.B. (Afiliation, City, State, Country); Author 2, C. (Afiliation, City, State, Country). Phase describing the material, year. (phase: Personal communication; Private communication; Unpublished work; etc.)

 Conference Proceedings:

17. Author 1, A.B.; Author 2, C.D.; Author 3, E.F. Title of Presentation. In *Title of the Collected Work* (if available), Proceedings of the Name of the Conference, Location of Conference, Country, Date of Conference; Editor 1, Editor 2, Eds. (if available); Publisher: City, Country, Year (if available); Abstract Number (optional), Pagination (optional).

 Thesis:
18. Author 1, A.B. Title of Thesis. Level of Thesis, Degree-Granting University, Location of University, Date of Completion.

 Websites:
19. Title of Site. Available online: URL (accessed on Day Month Year).
Unlike published works, websites may change over time or disappear, so we encourage you create an archive of the cited website using a service such as **WebCite**. Archived websites should be cited using the link provided as follows:
20. Title of Site. URL (archived on Day Month Year).

RESEARCH REPORT

Title:

The Prevalence of Arrhythmias in Acute ST-Segment Myocardial Infarction in a Tertiary Academic Hospital in Johannesburg

Authors:

Mothusi Koketso Setshego Bsc (UB) MBBCh (UKZN), FCP (SA)

Anupa Patel MBBCh (Wits), FCP(SA), Cert.Cardiology(SA)

Dineo Mpanya MBChB (UCT), FCNP (SA), MMed (Wits), PhD (Wits)

Nqoba Tsabedze MBBCh (Wits), FCP(SA), Cert Cardio (SA), MMed (Wits)

Institutional address:

Division of Cardiology, Department of Internal Medicine, School of Clinical Medicine, Faculty of Health Sciences, University of the Witwatersrand, Parktown, Johannesburg,

Corresponding author:

Mothusi Koketso Setshego, Division of Cardiology, Department of Internal Medicine, School of Clinical Medicine, Faculty of Health Sciences, University of the Witwatersrand, Parktown, Johannesburg, Gauteng, 2193, South Africa. A0067294@wits.ac.za

Abstract

South Africa has a rising prevalence of ischaemic heart diseases. Patients with acute myocardial infarction who succumb to sudden cardiac death tend to have fatal arrhythmias. This study aims to determine the prevalence of arrhythmias in patients with acute ST-segment elevation myocardial infarction (STEMI). We retrospectively reviewed medical records of patients with STEMI at a tertiary academic hospital for a 5 year period between 1 January 2015 and 31 December 2019. The study population comprised 259 patients with a mean age of 56.0 ±10.8 years. Dyslipidaemia was the most common co-morbidity, found in 169 (65.2%) patients. There were 125 (48.3%), 123 (47.5%) and 11 (4.2%) patients with inferior, antero-septal and lateral myocardial infarction, respectively. The most common arrhythmia was a sinus tachycardia, found in 80 (12.1%) patients, followed by a sinus bradycardia in 37 (5.6%). Thirty-seven of the 80 (70.1%) patients with a sinus tachycardia had ST-elevation in the anterior leads, and 19(51.3%) with a sinus bradycardia had ST-elevation in the inferior leads ($p<0.009$). Among the 143 patients with left anterior descending artery stenosis, 72 (50.3%) had a sinus tachycardia, while 19 (13.3%) had a sinus bradycardia ($p<0.001$). The most common arrhythmias in STEMI patients were a sinus tachycardia and bradycardia, accounting for arrhythmias in 36 and 27% of all patients, respectively.

Conclusion:The most common arrhythmias in ST-elevation MI patients were a sinus tachycardia and bradycardia, accounting for arrhythmias in 36% and 27% of all patients, respectively. Future prospective and multicentre studies are required in low-and-middle-income countries, which will determine the true burden of arrhythmia in acute STEMI.

Keywords: myocardial infarction; electrocardiogram; arrhythmia

Introduction

Cardiovascular diseases are responsible for approximately 17 million deaths globally [1].Myocardial infarction (MI) leads to significant morbidity and mortality [1]. Those who survive the initial insult may experience life-threatening arrhythmias. Patients with acute MI who succumb to sudden cardiac death have been found to have underlying fatal arrhythmia [2]. In sub-Saharan Africa (SSA), there is an increasing burden of cardiac arrhythmias [3, 4]. The mainstay of treatment for arrhythmias has been anti-arrhythmic drugs. Several clinical trials have evaluated whether acute or chronic anti-arrhythmic drugs can reduce mortality in post-MI patients. Of these, acute and long-term beta-blockers, independently and in combination, had been shown to reduce mortality.

Cardiac arrhythmias are common in patients with ST-segment elevation myocardial infarction (STEMI), with a prevalence and mortality rate expected to rise by 70% and 74% by 2030 in African men and women respectively [4, 5] and frequently occur early after the development of myocardial infarction, and can lead to sudden cardiac death [5, 6]. Advances in modern antithrombotic therapy, anti-arrhythmic therapy, primary percutaneous coronary intervention (PCI), and secondary prevention treatments have led to declining mortality rates [7-12]. Our study aimed to estimate the prevalence rate of arrhythmias in acute STEMI patients hospitalised in a tertiary academic hospital in Johannesburg, South Africa

Materials and Methods

Study Design and Participants

We retrospectively reviewed inpatient medical records of consecutive patients with STEMI who presented to the Charlotte Maxeke Johannesburg Academic Hospital (CMJAH) in the Division of Cardiology between 1 January 2015 and 31 December 2019. The CMJAH is a tertiary state-owned academic hospital in Johannesburg, South Africa that receives patients referred from Helen Joseph Hospital, which is part of the cluster of academic hospitals in the University of the Witwatersrand. The Division of Cardiology at the CMJAH is equipped with an intensive cardiac care unit, cardiac wards, angiography suite, electrophysiology laboratory and outpatient services. All patients 18 years and older with a diagnosis of STEMI as evidenced by electrocardiogram (ECG) changes, clinical profile and/or diagnostic angiography were included in the study. Patients with Q-waves and acute coronary syndromes (ACS) other than STEMI were excluded from the analysis.

2.2. Data Collection

Data were collected from the electronic health record system (EHR) that houses data of all patients admitted in the cardiology wards. Furthermore, coronary angiogram reports, images, and ECG printouts were reviewed when available. The extracted data included demographic data, co-morbidities, vital signs, laboratory parameters, coronary angiography findings, and discharge medications. A tachycardia was defined as a heart rate of above 100 beats per minute, and a heart rate of less than 60 beats per minute was used to define a bradycardia. The outcome in-hospital all-cause mortality was also obtained from the patient status documented on the EHR upon discharge

from the cardiac wards. Approval to conduct the study was granted by the University of the Witwatersrand Human Research Ethics Committee (certificate number: M210521) and relevant hospital authorities. The study adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [13].

Statistical Analysis

Categorical variables are expressed as frequencies and percentages and were compared using the Pearson Chi-square test. Continuous variables with a normal and non-normal distribution are expressed as the mean and standard deviation, and the median and interquartile range (IQR), respectively. The Student's t-test was used to compare means of continuous variables with a normal distribution, while continuous variables with a non-normal distribution were tested using the Wilcoxon rank sum (Mann–Whitney) test. Analysis of variance (ANOVA) was conducted to test the equality of the mean in three or more groups, and post hoc analysis of variables with statistically significant p-values on the ANOVA was done using the Tukeys' test. To compare continous variables with a non-normal distribution in three or more groups, a Kruskal-Wallis equality of the population rank test was used. Univariable and multivariable logistic regression analyses were done to identify predictors of arrhythmias. Thereafter, variables with a p-value less than 0.05 on univariable logistic regression analysis were selected and included in the multivariable logistic regression model. Confidence intervals were set at 95% (95% CI) and a p-value of less than 0.05 represents statistical significance. Analyses were conducted using Stata version 17.0 (StataCorp, College Station, TX, USA).

Results

Baseline demographic and clinical characteristics of patients with acute ST-segment elevation myocardial infarction

The final study population comprised 659 patients with acute STEMI, of which 203 (30.8%) had concomitant arrhythmias on the baseline ECG (Figure 1). Among the 659 patients with STEMI, the mean age was 56.0 ± 11.4 years and 518 (78.6%) patients hospitalised with an acute STEMI were males. There were 268 (40.7%), 266 (40.4%) and 125 (19.0%) patients with inferior, anterior or septal and lateral MI, respectively.

Smoking was the most common risk factor for atherosclerotic cardiovascular diseases (ASCVD), reported in 370 (56.2%) patients (Table 1). Diabetes mellitus was documented in 185 (28.1%) patients, and diabetic patients had a median glycated haemoglobin (HbA1c) of 8.7% (IQR: 7.3 – 10.5). The HbA1c did not differ significantly in diabetic patients with and without arrhythmias. The mean systolic and diastolic blood pressure were 124 ± 26.1 and 78 ± 11.4 mmHg, respectively, and the median heart rate on the baseline ECG was 83 (IQR: 72 – 95) beats per minute (bpm).

Arrhythmias in patients with acute ST-segment elevation myocardial infarction

The most common arrhythmia was a sinus tachycardia, found in 80 (12.1%) of patients with STEMI, followed by a sinus bradycardia in 37 (5.6%), and right bundle branch block in 23 (3.5%) (Figure 2). Thirty-seven of the 80 (46.2%) patients with a sinus tachycardia had ST-segment elevation in the anterior leads. There were 37 patients with STEMI and a bradycardia, of which 19 (51.3%) had ST elevation in the inferior leads. Among the 17 patients with complete heart block, 13 (76.5) of these patients had an inferior MI (p=0.009). Intraventricular blocks, left bundle branch (LBBB) and right bundle branch (RBBB) were found in 17 (2.6%) and 23 (3.5%) patients with STEMI, respectively, and 7 (41.2%) patients with LBBB had an inferior MI.

The median baseline troponin level at the time of hospitalisation was 3623 ng/l (1496 – 7193), and it did not differ between patients with and without arrhythmias [3716 (IQR: 1587 – 8808) vs 3623 (1397 – 6810), p=0.226]. Patients with STEMI had a median creatine kinase MB mass of 87.1 µg/l (IQR: 34.0 – 231.8), and there were no statistically significant differences in serum creatine kinase MB levels in patients with and without arrhythmias [69.8 µg/l (IQR: 35.2 – 207.2) vs 91.5 µg/l (IQR: 20.0 – 248.7), p=0.927).

Arrhythmias and diseased vessels on coronary angiography

Coronary angiography revealed single, double and triple vessel disease in 114 (56.2%), 48 (23.6%), and 30 (14.8%) patients with STEMI and concomitant arrhythmias. There were four (2.0%) patients without evidence of obstructive CAD on coronary angiography, and angiographic data was not available in seven (3.4%) patients. Among the 203 patients with arrhythmias, 119 (58.6%) had stenosis in the left anterior descending artery (LAD), 109 (53.7%) in the right coronary artery (RCA), 56 (27.6%) in the circumflex artery, and four (2.0%) in the left main coronary artery. A sinus tachycardia was found in 55 (46.2%) of the 119 patients with LAD lesions. Sixteen (13.4%) patients with stenosis in the LAD artery had a sinus bradycardia, and 13 (11.0%) had a right bundle branch block (p=0.061). Overall, there was no association between angiographic disease and arrhythmias.

In-hospital management of patients with ST-segment elevation myocardial infarction and arrhythmias

A pharmaco-invasive management strategy (*thrombolytic therapy and PCI)* was implemented in 76 (37.4%) of the 203 patients with STEMI and arrhythmias. Fifty-one (25.1%) patients with STEMI and arrhythmias were treated with thrombolytic therapy only (*Streptokinase or Alteplase)*, while 37 (18.2%) patients had primary or ad hoc PCI without thrombolytic therapy. The rest of the 39 (19.2%) patients with STEMI and arrhythmias were managed conservatively. There was no association between the management strategies and the presence of arrhythmias.

The median duration between the onset of chest pain and catheterisation was two days (IQR: 1 - 3), and it did not differ significantly in patients with and without arrhythmias [1 day (IQR: 1 – 3) vs 2 days (IQR: 1 – 3), p=0.065]. Fifty-six (27.6%) patients had PCI to the LAD artery, 48 (23.6%) had PCI to the RCA, 15 (7.4%) had PCI to the circumflex artery, 5 (2.5%) had PCI to the obtuse marginal or ramus branch and one (0.5%) patient had PCI to the left main coronary artery. Seven patients had interventions performed in the LAD artery and RCA, while in one patient, PCI was performed in the LAD and circumflex arteries

Upon discharge from the hospital, 502 (76.2%) patients with STEMI were prescribed oral beta-blockers. Of these 502 patients on beta blockers, 129 (25.7%) had arrhythmias (p<0.001). Angiotensin-converting enzyme (ACE) inhibitors were prescribed in 422 (64.0%) patients, and 102 (24.2%) of these patients had arrhythmias (p<0.001). Sixty-seven (10.2%) patients with STEMI were on mineralocorticoid antagonists. In the entire study population, 19 of the 659 (2.9%) patients with STEMI were referred for pacemaker implantation before discharge from the hospital (p<0.001). Of these patients referred for pacemaker implantation, 17 (89.5%) had STEMI and underlying arrhythmias.

Predictors of arrhythmias, occurrence of complications and mortality outcomes in all patients with ST-elevation myocardial infarction

Among 659 patients with STEMI, 47 (7.1%) had cardiogenic shock, and 29 (61.7%) were treated with inotropes (p<0.001). In the 39 patients with haemodynamic instability, 28 (71.8%) had arrhythmias (p<0.001). The median duration of hospitalisation was 3.5 days (2.0 – 6.0), and it did not differ in patients with and without arrhythmias. The in-hospital all-cause mortality rate in the entire study population was 6.4% (95% CI: 4.6 – 8.5). Thirty-five (83.3%) of the 42 non-survivors had arrhythmias. A preserved eGFR [OR: 0.99 (95% CI: 0.98 – 0.99), p=0.026] and haemodynamic instability [OR: 3.33 (95% CI: 1.43 – 7.79), p=0.005] at the time of hospitalisation emerged as significant predictors of experiencing arrhythmias (Table 3).

Discussion

We conducted a retrospective study on 659 patients hospitalised with acute STEMI in a tertiary academic hospital in Johannesburg, South Africa. Among these patients, 30.8% had concomitant arrhythmias. The most prevalent arrhythmia was a sinus tachycardia, found in 12.1% of patients with STEMI, followed by a sinus bradycardia in 5.6% of patients. Shah et al. studied 110 patients hospitalised with acute MI in a tertiary care cardiac centre in Pakistan and found a sinus tachycardia in 36.4% of patients [14]. Also, Sallman found a sinus tachycardia in 38.8% of patients with an inferior MI treated in Iraq [15]. A sinus tachycardia in the seting of acute MI is a non-specific finding that may reflect a catecholamine surge, pain, anxiety, haemorrhage, or heart failure [16]. Considering that a sinus tachycardia is characterised as a non-lethal arrhythmia, there are limited or no contemporary research studies reporting on survival rates in acute STEMI patients with this arrhythmia.

In our study, 5.6% of patients hospitalised with STEMI had a sinus bradyarrhythmia. However, Adgey and colleagues, who found a sinus or nodal bradycardia in 27.0% of patients with infarcted hearts within the first hour after acute MI [17]. A sinus bradyarrhythmia is common in patients with inferior or posterior acute MI, since in approximately 60% of people, the right coronary supplies the sinoatrial node [15]. A sinus bradycardia is typically observed in the first 1-2 hours after acute MI. In the early phases of an acute MI, the resultant sinus bradycardia may be protective. The mechanism behind a sinus bradycardia is likely related to cardiac vagal stimulation. When emergency treatment is required for a symptomatic bradycardia, intravenous atropine remains the first-line pharmacological therapy, and transcutaneous or temporary pacing is usually indicated if the patient fails to respond to atropine [11-15].

We found that patients with STEMI had a mean age of 56 years and were predominantly Caucasian. Smoking was the most prevalent risk factor for ASCVD, reported in 56.2%, followed by hypertension in 53.1% and dyslipidaemia in 30% of patients with STEMI [18]. Although cigarette smoking is associated with multiple chronic illnesses such as ASCVD, chronic obstructive pulmonary diseases, and diabetes mellitus, the underlying pathophysiological pathway leading to ASCVD is not well described [19, 20]. Tobacco smoking likely induces inflammation, thrombosis, immune system dysregulation and oxidative stress in the coronary vessels, leading to endothelial cell damage in the smooth muscle cells lining the coronary arteries [21]. Furthermore, cigarette smoke results in higher serum total cholesterol, triglycerides, very low density, low-density lipoprotein cholesterol (LDL-C) levels, and lower high-density lipoprotein cholesterol levels [22] .

In our study, more than half of patients had hypertension. This condition is common in SSA, and it remains poorly diagnosed and controlled.

We also found that 40.1% of STEMI patients in our study had dyslipidaemia. Dyslipidaemia is another well-established risk factor for ASCVD [26-28]. This finding supports the need to aggressively treat lipid abnormalities, primarily in patients with established cardiovascular risk factors. Low-density lipoprotein cholesterol is the primary driver of atherogenesis that leads to plaque formation and a subsequent decrease in the size of the coronary lumen, hence increasing myocardial oxygen demand [29, 30]. Lipid levels should be measured in every patient with acute MI, and statin therapy should be initiated to achieve LDL-C serum levels of less than 1.8 mmol/l [3]. The demographic parameters such as age, gender and ethnicity did not differ between patients with and without arrhythmias. However, patients with arrhythmias had hypotension and were in advanced stages of heart failure. These patients were more likely to complicate with cardiogenic shock. The serum potassium levels were slightly higher in patients with arrhythmias, and the renal function was impaired. Patients with arrhythmias who were active smokers were likely to present with an inferior MI.

A bundle branch block during acute MI is associated with a mortality rate of 13.2% at 30 days [21,22]. Conduction from the His bundle is transmitted through three fascicles, the anterior division of the left bundle, the posterior division of the left bundle, and the right bundle. An abnormality of electrical conduction through any of these bundles causes a bundle branch block. Bundle branch blocks were uncommon in our study. We found RBBB and LBBB in 3.5% and 2.6% of STEMI patients, respectively. The right bundle branch receives its dominant blood supply from the LAD artery [31]. Therefore, a new RBBB, usually observed in 42% of patients with acute MI, suggests a large infarct territory [22-24]. However, progression to complete AV block is uncommon. In patients who develop an anterior MI and a new RBBB, the substantial risk of death is mostly from cardiogenic shock, most likely due to the large infarct size.

Only 1.1% of STEMI patients in our study had a ventricular tachycardia (VT). A non-sustained VT that occurs immediately after the peri-infarction period is independently associated with an increased risk of death and other cardiovascular outcomes, including cerebrovascular accidents [26-27]. Sustained VT, defined as three or more ventricular ectopic beats at a rate greater than 100 bpm lasting more than 30 seconds, is associated with an in-hospital mortality rate of 20.3% [25]. Prescribing beta-blockers in the first 24 hours of acute MI reduces the in-hospital mortality rate without worsening heart failure. Amiodarone has no added benefit over concomitant beta-blocker therapy [25,26]. However, emergency treatment for sustained VT is mandatory because VT can progress to ventricular fibrillation.

Among the 659 STEMI patients studied, 1.5% had atrial fibrillation. Atrial fibrillation during AMI is associated with an increased risk of mortality, with a crude mortality rate of about 60.7 per 1000 person-years [9]. Atrial fibrillation is a chaotic, irregular atrial rhythm at a rate of 300-600 beats per minute. The risk of new-onset atrial fibrillation is high in the first two months after acute MI and decreases by the twelfth month [10]. Documenting and analysing atrial fibrillation as an underlying co-morbidity in patients presenting with STEMI would have enabled us to identify patients with acute onset atrial fibrillation. Although not considered a lethal arrhythmia, atrial fibrillation may accelerate the progression of ventricular dysfunction, such as in patients with ischaemic cardiomyopathy. Early and effective management of atrial fibrillation is challenging in most LMICs, largely because of the limited access to facilities with capacity to monitor the International Normalised Ratio in patients taking oral warfarin.

The in-hospital all-cause mortality rate among acute STEMI patients was 6.4% (95% CI: 4.6 – 8.5). The mortality rate in STEMI patients with underlying arrhythmias is poorly documented and possibly influenced by the most prevalent arrhythmia in the cohort studied. For example, the in-hospital all-cause mortality rate in STEMI patients with and without arrhythmias has been reported to be 15.5%, and the most common arrhythmia in this study by Shah was an accelerated idioventricular rhythm, seen in 37.3% of all STEMI patients [14]. We found that 83.3% of non-survivors had underlying arrhythmias. We also explored clinical parameters associated with the presence of arrhythmias and found that renal dysfunction predisposed patients to be diagnosed with arrhythmias. Patients with chronic kidney disease are susceptible to experiencing cardiac arrhythmias, most commonly atrial fibrillation with a prevalence of 16 – 21% in non-dialysed patients and 15 – 40% in patients on dialysis [32]. Furthermore, we found that haemodynamically unstable patients were three times more likely to have arrhythmias on the ECG. These patients may have also been clinically unstable due to underlying arrhythmias. While collecting data, haemodynamic instability was defined as a mean arterial pressure less than 65 mmHg or a documentation of unrecordable blood pressure or use of inotropes. Thirty-nine patients were haemodynamically unstable, and 71.8% had underlying arrhythmias.

Newer first-generation wearable device therapy for remote monitoring and diagnosis of arrhythmias would greatly benefit low-and-middle-income countries, considering the increasing burden of cardiovascular diseases. The added benefit of these devices would be the early detection of life-threatening arrhythmias, hence facilitating the early implementation of appropriate interventions that further decrease the morbidity and mortality associated with arrhythmias in acute STEMI.

Our study has several limitations. This was a single-centre study with a relatively small sample size. In addition, we exclude a significant number of STEMI patients with missing pertinent clinical data, such as a definite diagnosis confirming STEMI or coronary angiography findings. Some of these patients were initially managed by referring hospitals, so we could not access their medical records. Arrhythmia-specific therapeutic strategies such as oral amiodarone, chemical cardioversion and direct current shock were not well documented and subsequently not analysed as part of the study. Although available, important clinical parameters such as the left ventricular ejection fraction were not collected. Ideally, a prospective study with electrocardiograms done multiple times would have offered more insights into the burden of arrhythmias in STEMI. Nevertheless, despite these limitations, we could describe common arrhythmias that should be anticipated and potentially prevented in patients with acute STEMI.

Conclusions

The most common arrhythmias in ST-elevation MI patients were a sinus tachycardia and bradycardia, accounting for arrhythmias in 36% and 27% of all patients, respectively. Future prospective and multicentre studies are required in low-and-middle-income countries, which will determine the true burden of arrhythmia in acute STEMI.

Abbreviations

AIVR: Accelerated idioventricular rhythm

AMI: Acute myocardial infarction

AF: Atrial fibrillation

AV: Atrioventricular

LAD: Left anterior descending

LAFB: Left anterior fascicular block

LBBB: Left bundle branch block

MI: Myocardial infarction

PCI: Percutaneous coronary intervention

PVC: Premature ventricular contraction

RBBB: Right bundle branch block

RV: Right ventricle/Right ventricular

STEMI: ST-segment elevation myocardial infarction

VA: Ventricular arrhythmia

VT: Ventricular tachycardia

VF: Ventricular fibrillation

DECLARATIONS

Ethics approval and consent to participate

The study complied with the Declaration of Helsinki. Permission to conduct the study was granted by the University of the Witwatersrand Human Research Ethics Committee (Medical) (Certificate number: M210521). Patients were not required to give informed consent as this was a retrospective study.

Supplementary Materials

None

Author Contributions

Conceptualisation, M.S. and N.T.; methodology, M.S.,A.P.,D.M and N.T; formal analysis, D.M. and M.S; investigation, M.S.; data curation, M.S.; writing—original draft preparation, M.S.; writing—review and editing, M.S.,A.P.,D.M and N.T; visualisation, M.S.,A.P.,D.M and N.T.; supervision, A.P.,D.M and N.T. All authors have read and agreed to the published version of the manuscript.

Funding

This research received no external funding.

Data Availability Statement

Data is available at a reasonable request from the corresponding author.

Acknowledgements

The authors would like to thank Dr Samantha Nel for assisting with retrieving the medical records of the study patients.

Conflicts of Interest

NT is a cardiologist and has received consultation fees from Acino Health Care Group, AstraZeneca, Boston Scientific, Novartis Pharmaceuticals, Novo Nordisk, Pfizer, Sanofi, Phillips, Servier, Takeda, and Merck. He has also received educational and travel grants from Medtronic, Biotronik, Boston Scientific and Vertice Health Care Group. None of the other authors have any relevant financial or professional disclosures.

REFERENCES

[1] Mendis S, Puska P, et al., Global atlas on cardiovascular disease prevention and control. Geneva: World Health Organization in collaboration with the World Heart Federation and the World Stroke Organization; 2011;5-6.

[2] Eckart RE, Shry EA, et al., Sudden death in young adults: an autopsy-based series of a population undergoing active surveillance. J Am Coll Cardiol. 2011;58:1254-61. https://doi.org/10.1016/j.jacc.2011.01.049

[3] Bahiru E, Temu T, et al., Presentation, management and outcomes of acute coronary syndrome: a registry study from Kenyatta National Hospital in Nairobi, Kenya. Cardiovasc J Afr. 2018;29:225-30. https://doi.org/10.5830/CVJA-2018-017

[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;23:365-70. https://doi.org/10.5830/CVJA-2012-017

[5] Mandelzweig L, Battler A, et al., The second Euro Heart Survey on acute coronary syndromes: Characteristics, treatment, and outcome of patients with ACS in Europe and the Mediterranean Basin in 2004. Eur Heart J. 2006;27:2285-93. https://doi.org/10.1093/eurheartj/ehl196

[6] Mensah GA, Roth GA, 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:S6-10. https://doi.org/10.5830/CVJA-2015-036

[7] Benjamin EJ, Wolf PA, et al., Impact of atrial fibrillation on the risk of death: the Framingham Heart Study. Circulation. 1998;98:946-52. https://doi.org/10.1161/01.cir.98.10.946

[8] Fuster V, Rydén LE, et al., ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation: Executive Summary A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to Develop Guidelines for the Management of Patients With Atrial Fibrillation) Developed in Collaboration With the North American Society of Pacing and Electrophysiology. Circulation. 2001;104:2118-50.

[9] Ibanez B, James S, et al., 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Kardiol Pol. 2018;76:229-313. https://doi.org/10.5603/KP.2018.0041

[10] January CT, Wann LS, et al., 2019 AHA/ACC/HRS focused update of the 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Heart Rhythm. 2019;16:e66-e93. https://doi.org/10.1016/j.hrthm.2019.01.024

[11] Jernberg T, Johanson P, et al., Association between adoption of evidence-based treatment and survival for patients with ST-elevation myocardial infarction. JAMA. 2011;305:1677-84. https://doi.org/10.1001/jama.2011.522

[12] López-Sendón J, Swedberg K, et al., Expert consensus document on beta-adrenergic receptor blockers. Eur Heart J. 2004;25:1341-62. https://doi.org/10.1016/j.ehj.2004.06.002

[13] Shah JA, Naz F, et al., Incidence of Cardiac Arrhythmias in Acute Myocardial Infarction Patients Undergoing Primary Percutaneous Coronary Intervention and Associated Outcomes During the First 24 Hours. Cureus. 2021;13:e12599. https://doi.org/10.7759/cureus.12599

[14] Sallman OS, Complications and mortality in hospitalised patients with acute inferior myocardial infarction with or without right ventricular involvement. Tikrit Medical Journal. 2009;15:140-6.

[15] Frampton J, Ortengren AR, et al., Arrhythmias After Acute Myocardial Infarction. Yale J Biol Med. 2023;96:83-94. https://doi.org/10.59249/LSWK8578

[16] Lin J, Kristanto W, et al., Pulmonary embolism as a cause of unexplained sinus tachycardia after right ventricular myocardial infarction. Singapore Med J. 2013;54:e199-200. https://doi.org/10.11622/smedj.2013191

[17] Adgey AA, Geddes JS, et al., Incidence, significance, and management of early bradyarrhythmia complicating acute myocardial infarction. Lancet. 1968;2:1097-101. https://doi.org/10.1016/s0140-6736(68)91577-8

[18] Pol T, Held C, et al., Dyslipidemia and Risk of Cardiovascular Events in Patients With Atrial Fibrillation Treated With Oral Anticoagulation Therapy: Insights From the ARISTOTLE (Apixaban for Reduction in Stroke and Other Thromboembolic Events in Atrial Fibrillation) Trial. J Am Heart Assoc. 2018;6-7. https://doi.org/10.1161/JAHA.117.007444

[19] Balling M, Nordestgaard BG, et al., Small Dense Low-Density Lipoprotein Cholesterol Predicts Atherosclerotic Cardiovascular Disease in the Copenhagen General Population Study. J Am Coll Cardiol. 2020;75:2873-5. https://doi.org/10.1016/j.jacc.2020.03.072

[20] Lu Y, Cui X, et al., The Functional Role of Lipoproteins in Atherosclerosis: Novel Directions for Diagnosis and Targeting Therapy. Aging Dis. 2022;13:491-520. https://doi.org/10.14336/AD.2021.0929

[21] Boren J, Chapman MJ, et al., Low-density lipoproteins cause atherosclerotic cardiovascular disease: pathophysiological, genetic, and therapeutic insights: a consensus statement from the European Atherosclerosis Society Consensus Panel. Eur Heart J. 2020;41:2313-30. https://doi.org/10.1093/eurheartj/ehz962

[22] Gaggini M, Gorini F, et al., Lipids in Atherosclerosis: Pathophysiology and the Role of Calculated Lipid Indices in Assessing Cardiovascular Risk in Patients with Hyperlipidemia. Int J Mol Sci. 2022;20-24. https://doi.org/10.3390/ijms24010075

[23] Futami C, Tanuma K, et al., The arterial blood supply of the conducting system in normal human hearts. Surg Radiol Anat. 2003;25:42-9. https://doi.org/10.1007/s00276-002-0085-7

APPENDICES

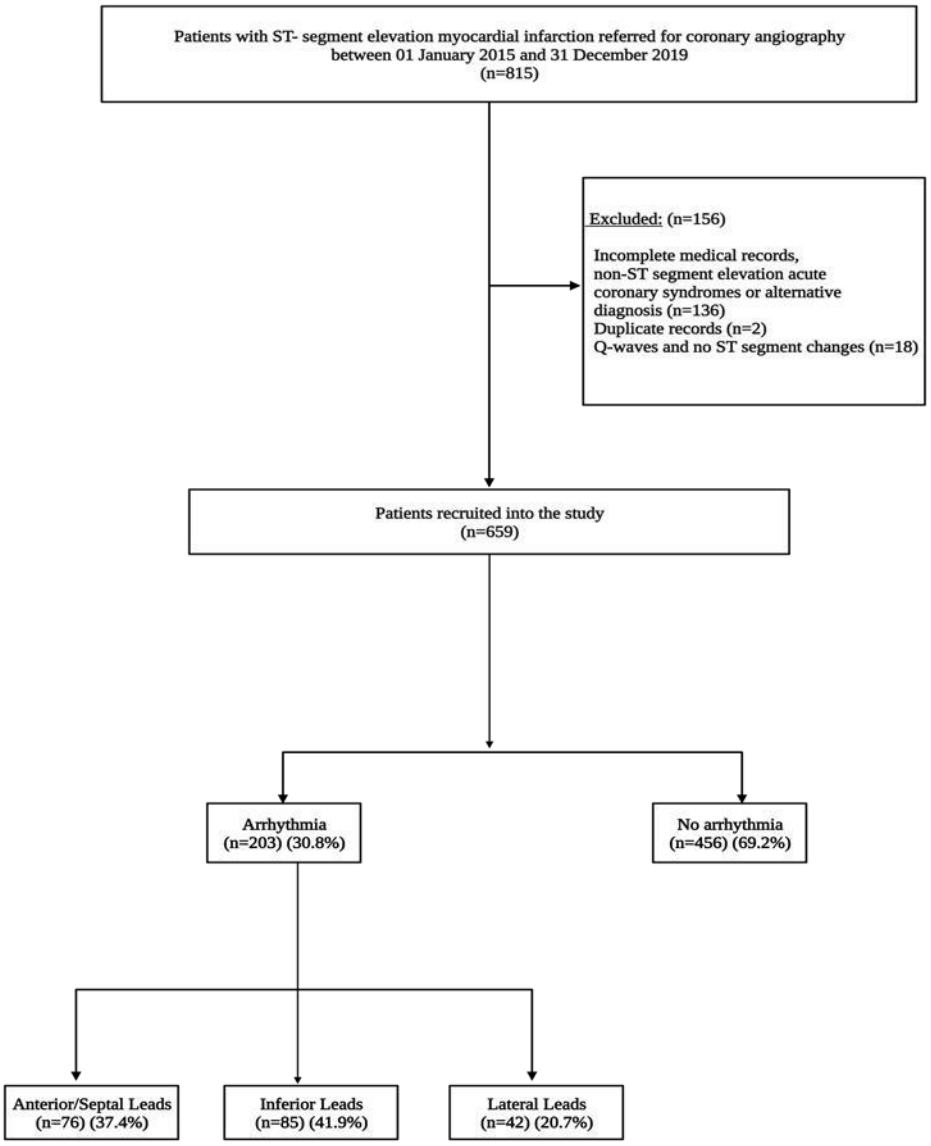


Figure 1. Study flow chart showing patient selection into the study. STEMI: ST-elevation myocardial infarction.

Table 1

		Arrhythmia		
	All Patients n=659	**Yes n=203 (30.8%)**	**No n= 456 (69.2%)**	**p-value**
Age ,years	56.1 ± 11.4	57.2 ± 11.8	55.5 ± 11.2	0.087
Male	518 (78.6)	159 (78.3)	359 (78.7)	0.907
Ethnicity				0.701
Caucasian	310 (47.0)	91 (44.8)	219 (48.0)	
African	161 (24.4)	51 (25.1)	110 (24.1)	
Asian	141 (21.4)	49 (24.1)	92 (20.2)	
Mixed Ancestry	37 (5.6)	10 (4.9)	27 (5.9)	
Other	10 (1.5)	2 (1.0)	8 (1.7%)	
Risk factors for CAD				
Current Smoker	370 (56.2)	107 (52.7)	263 (57.8)	0.224
Hypertension	350 (53.1)	108 (53.2)	242 (53.1)	0.975
Dyslipidaemia	264 (40.1)	79 (38.9)	185 (40.6)	0.689
Diabetes mellitus	185 (28.1)	61 (30.0)	124 (27.1)	0.451
Previous smoker	55 (8.3)	20 (9.8)	35 (7.7)	0.351
Vital signs				
Heart rate, bpm	83 (72 – 95)	102 (57 – 115)	81 (73 – 88)	<0.001
Systolic BP	124 ± 26.1	119 ± 30.3	126 ± 23.8	0.002
Diastolic BP	78 ± 18.5	75 ± 20.8	80 ± 17.2	<0.001
NYHA Functional Class 1	584 (88.6)	165 (81.3)	419 (91.9)	<0.001

2	36 (5.5)	13 (6.4)	23 (5.0)	0.478
3	17 (2.6)	7 (3.5)	10 (2.2)	0.348
4	22 (3.3)	18 (8.9)	4 (0.9)	<0.001
Killip Class				
1	559 (84.8)	149 (73.4)	410 (89.9)	<0.001
2	29 (4.4)	11 (5.4)	18 (3.9)	0.395
3	24 (3.6)	14 (6.9)	10 (2.2)	0.003
4	47 (7.1)	29 (14.3)	18 (3.9)	<0.001
Biochemical Investigations				
Haemoglobin, g/dl	14.3 ± 2.2	14.1 ± 2.4	14.4 ± 2.1	0.064
Sodium, mmol/l	140 (137 – 142)	139 (136 – 141)	140 (137 – 142)	0.067
Potassium, mmol/l	4.2 (3.9 – 4.6)	4.3 (4.0 – 4.7)	4.2 (3.9 – 4.5)	0.002
Urea, mmol/l	5.6 (4.5 – 7.4)	6.3 (4.8 – 8.1)	5.3 (4.2 – 6.9)	<0.001
Creatinine, µmol/l	87 (73 – 107)	98 (77 – 121)	84 (72 – 100)	<0.001
eGFR, ml/min/1.73m²	76.9 (60.5 – 94.6)	68.1 (48.7 – 89.8)	80.4 (64.7 – 96.3)	<0.001
Total cholesterol	4.6 ± 1.4	4.6 ± 1.6	4.7 ± 1.3	0.946
Triglycerides	1.4 (0.9 – 2.2)	1.3 (0.9 – 2.1)	1.4 (0.9 – 2.2)	0.487
LDL	3.0 (2.1 – 3.7)	2.9 (2.1 – 3.6)	3.0 (2.1 – 3.7)	0.582
HDL	1.0 (0.8 – 1.3)	0.9 (0.8 – 1.2)	1.0 (0.8 – 1.3)	0.137

Categorical data is expressed as frequencies and percentages. Data is represented as the mean and standard deviation (±) for continuous features with a normal distribution and as the median and interquartile ranges (p25-p75) when the distribution is non-normal. BP: blood pressure; CAD: coronary artery disease; eGFR: estimate glomerular filtration rate; HDL: high-density lipoprotein; LDL: low-density lipoprotein; NYHA: New York Heart Association.

Figure 2

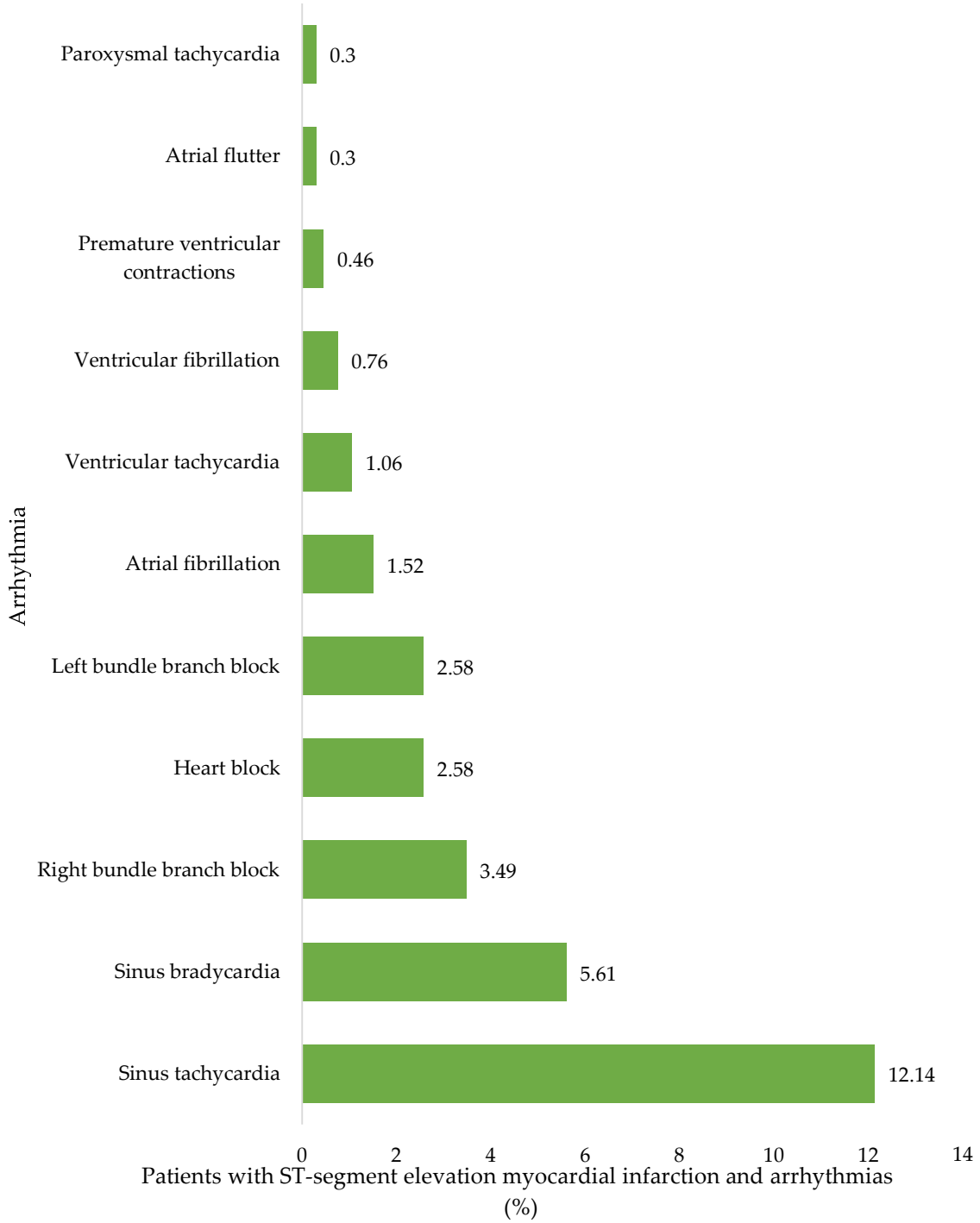


Figure 2. Bar graph showing arrhythmias in patients with ST-elevation myocardial infarction.

Table 2. Baseline demographic and clinical characteristics of patients with acute ST-elevation myocardial infarction and arrhythmias stratified according to the lead territory on the electrocardiogram

		Lead Territories			
	All patients with STEMI & arrhythmias n=203	**Anterior or Septal n=76 (28.5%)**	**Inferior n=85 (31.7%)**	**Lateral n= 42 (33.6%)**	**p-value**
Age (years)	57.2 ± 11.8	56.0 ± 11.8	57.5 ± 12.4	58.8 ± 10.8	0.626
Male	159 (78.3)	62 (81.6)	63 (74.1)	34 (80.9)	0.465
Ethnicity					0.068
Caucasian	91 (44.8)	28 (36.8)	47 (55.3)	16 (38.1)	
African Ancestry	51 (25.1)	28 (36.8)	11 (12.9)	12 (28.5)	
Asian	49 (24.1)	15 (19.7)	22 (25.9)	12 (28.5)	
Mixed Ancestry	10 (4.9)	4 (5.2)	4 (4.7)	2 (4.8)	
Other	2 (1.0)	1 (1.3)	1 (1.2)	0 (0.0)	
Risk factors for CAD					
Hypertension	108 (53.2)	38 (50.0)	47 (55.3)	23 (54.7)	0.777
Smoker	107 (52.7)	33 (43.4)	55 (64.7)	19 (45.2)	0.014
Dyslipidaemia	79 (38.9)	29 (38.2)	31 (36.5)	19 (45.2)	0.626
Diabetes mellitus	61 (30.0)	27 (35.5)	18 (21.2)	16 (38.1)	0.062
Previous smoker	20 (9.8)	8 (10.5)	6 (7.1)	6 (14.3)	0.424
Previous CAD	5 (2.5)	1 (1.3)	3 (3.5)	1 (2.4)	0.664
Vital signs					
Heart rate, bpm	93 ± 36.1	101 ±31.3	85 ± 39.4	91 ± 34.8	0.125
Systolic BP	119 ±30.2	114 ±28.1	121 ±30.6	125 ± 32.1	0.580
Diastolic BP	75 ± 20.7	75 ± 17.7	71 ± 22.5	81 ± 20.9	0.108

NYHA Functional Class					
1	165 (81.3)	60 (78.9)	74 (87.1)	31 (73.8)	0.159
2	13 (6.4)	2 (2.6)	5 (5.9)	6 (14.3)	0.045
3	7 (3.4)	2 (2.6)	4 (4.7)	1 (2.4)	0.705
4	18 (8.9)	12 (15.8)	2 (2.3)	4 (9.5)	0.011
Killip Class					
1	149 (73.4)	52 (68.4)	68 (80.0)	29 (69.0)	0.195
2	11 (5.4)	2 (2.6)	5 (5.9)	4 (9.5)	0.277
3	14 (6.9)	8 (10.5)	2 (2.3)	4 (9.5)	0.093
4	29 (14.3)	14 (18.4)	10 (11.8)	5 (11.9)	0.428
Biochemical Investigations					
Haemoglobin, g/dl	14.1 ± 2.4	14.2 ± 2.4	14.0 ± 2.2	13.9 ± 2.5	0.695
Sodium, mmol/l	139 ± 4.5	138 ± 4.1	140 ± 4.1	138 ± 5.4	0.076
Potassium, mmol/l	4.3 ±0.6	4.5 ± 0.6	4.3 ± 0.7	4.1 ± 0.6	0.830
Urea, mmol/l	6.3 (4.8 – 8.1)	6.2 (4.8 – 9.0)	6.5 (4.9 – 8.0)	6.0 (4.7 – 7.8)	0.765
Creatinine, µmol/l	97 (77 – 121)	102 (82 – 126)	86 (72 – 115)	103 (80 – 130)	0.058
eGFR, ml/min/1.73m²	68.1 (48.7 – 89.8)	65.5 (49.3 – 86.1)	76.9 (54.3 – 92.4)	61.9 (45.6 – 89.0)	0.302
Total cholesterol	4.5 (3.7 – 5.4)	4.4 (3.5 – 5.0)	4.8 (4.0 – 5.7)	4.3 (3.4 – 5.2)	0.053
Triglycerides	1.4 (0.9 – 2.1)	1.3 (0.9 – 1.8)	1.7 (1.0 – 2.6)	1.2 (0.9 – 1.5)	0.149
LDL	2.9 (2.1 – 3.6)	2.8 (1.9 – 3.4)	3.1 (2.4 – 3.8)	2.8 (2.0 – 3.6)	0.225
HDL	1.0 (0.8 – 1.2)	1.0 (0.7 – 1.2)	0.9 (0.8 – 1.2)	1.0 (0.9 – 1.3)	0.271

Categorical data is expressed as frequencies and percentages. Data is represented as the mean and standard deviation (±) for continuous features with a normal distribution and as the median andinterquartile ranges (p25-p75) when the distribution is non-normal. BP: blood pressure; CAD: coronary artery disease; CVD: cardiovascular disease; eGFR: estimate glomerular filtration rate; HDL: high-density lipoprotein; LDL: low-density lipoprotein; NYHA: New York Heart Association; STEMI: ST-segment elevation myocardial infarction

Table 3. Univariable and multivariable logistic regression analysis for predictors of arrhythmias

	Univariable Logistic Regression		**Multivariable Logistic Regression**	
Variable	**OR (95% CI)**	**p-value**	**OR (95% CI)**	**p-value**
Age, years	1.01 (0.99 – 1.02)	0.088		
Chest pain to catheterisation time, days	0.99 (0.98 – 1.00)	0.791		
Systolic BP, mmHg	0.99 (0.98 – 0.99)	0.002	1.0 (0.99 – 1.01)	0.639
Diastolic BP, mmHg	0.98 (0.97 – 0.99)	0.001	0.99 (0.97 – 1.00)	0.236
Haemoglobin, g/dl	0.93 (086 – 1.00)	0.065		
Sodium, mmol/l	0.99 (0.97 – 1.01)	0.734	0.99 (0.97 – 1.01)	0.593
Potassium, mmol/l	1.45 (1.08 – 1.92)	0.011	1.07 (0.78 – 1.47)	0.656
Urea, µmol/l	1.09 (1.04 – 1.14)	<0.001	1.02 (0.99 – 1.06)	0.106
eGFR	0.98 (0.98 – 0.99)	<0.001	0.99 (0.98 – 0.99)	0.026
NYHA, class 1	0.38 (0.23 – 0.62)	< 0.001	1.96 (0.79 – 4.86)	0.147
NYHA, class 2	1.29 (0.64 – 2.59)	0.479		
NYHA, class 4	11.0 (3.67 – 32.9)	< 0.001	2.77 (0.70 – 10.95)	0.147
Killip, class 1	0.31 (0.20 – 0.47)	< 0.001	0.50 (0.18 – 1.39)	0.187
Killip, class 3	3.31 (1.44 – 7.57)	0.005	1.71 (0.45 – 6.40)	0.428
Killip, class 4	4.05 (2.19 – 7.49)	< 0.001	0.91 (0.27 – 3.01)	0.873
Dobutamine	4.38 (2.39 – 8.04)	< 0.001	2.08 (0.93 – 4.62)	0.073
Haemodynamic instability	6.47 (3.15 – 13.3)	< 0.001	3.33 (1.43 – 7.79)	0.005

BP: blood pressure; eGFR: estimate glomerular filtration rate; NYHA: New York Heart Association; OR: odds ratio. CI: confidence interval

F. PROTOCOL

Mothusi Koketso Setshego **Student number:2417975**
Staff number: A0067294

TITLE

THE PREVALENCE OF ARRHYTHMIAS IN ACUTE ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

Student: Mothusi Koketso Setshego

BSc (UB), MBChB (UKZN)

Primary Supervisor: Nqoba Tsabedze, Academic & Clinical Head – Division of Cardiology, Department of Internal Medicine, University of the Witwatersrand and the Charlotte Maxeke Johannesburg Academic Hospital

MBBCh (Wits), FCP(SA), MMed, Cert.Cardiology(SA)

Secondary Supervisor: Anupa Patel, Senior Consultant, Division of Cardiology, Department of Internal Medicine, University of the Witwatersrand and the Charlotte Maxeke Johannesburg Academic Hospital

MBBCh (Wits), FCP(SA), Cert.Cardiology(SA)

August 2020

TABLE OF CONTENTS

TITLE 1

INTRODUCTION 4

CLASSIFICATION OF PERI-INFARCT ARRHYTHMIAS 4

STUDY AIM 10

OBJECTIVES 10

RESEARCH METHOD 10

METHODOLOGY 10

STATISTICAL ANALYSIS 11

ETHICAL CONSIDERATION 11

TIMELINE 11

FUNDING 12

REFERENCES 12

APPENDIX 16

ABBREVIATIONS

1. AIVR: Accelerated idioventricular rhythm
2. AMI: Acute myocardial infarction
3. AF: Atrial fibrillation
4. AV: Atrioventricular
5. LAD: Left anterior descending
6. LAFB: Left anterior fascicular block
7. LBBB: Left bundle branch block
8. MI: Myocardial infarction
9. PCI: Percutaneous coronary intervention
10. PVC: Premature ventricular contraction
11. RBBB: Right bundle branch block
12. RV: Right ventricle/Right ventricular
13. STEMI: ST-segment elevation myocardial infarction
14. VA: Ventricular arrhythmia
15. VT: Ventricular tachycardia
16. VF: Ventricular fibrillation

INTRODUCTION

Cardiovascular disease is responsible for approximately 17 million deaths worldwide. [1] It is well established that myocardial infarction (MI) leads to significant morbidity and mortality. [1] Those who survive the initial insult can have complications, including but not limited to life-threatening arrhythmias. Patients with early myocardial infarction who succumb to sudden cardiac death generally have been found to have had a fatal arrhythmia. [2] An arrhythmia describes an abnormal heartbeat rhythm whereby the heart may beat too fast, too slow, too early, or irregularly.

The mainstay of treatment for arrhythmias has been anti-arrhythmic drugs. Several clinical trials have evaluated whether acute or chronic anti-arrhythmic drugs can reduce mortality in post-MI patients. Of these, acute and long-term beta-blockers, independently and in combination, had been shown to reduce mortality.[3]

Background and significance of the problem

In Sub-Saharan Africa, there is an increasing burden of cardiac arrhythmias.[4,5] Cardiac arrhythmias are common in patients with ST-elevation MI (STEMI), frequently occur early after the development of myocardial infarction, and can lead to sudden cardiac death.[6,7] Estimates from high-income countries indicate that the prevalence of in-hospital mortality varies between 6% -14%.[8] Still, in both developed countries and Sub-Saharan Africa, there is a lack of data regarding the prevalence of arrhythmias in acute STEMI.[8] Advances in modern antithrombotic therapy, primary percutaneous coronary intervention (PCI), and secondary prevention treatments have led to a fall in long-term mortality[9] but mortality rates in higher-risk patients remain high. Also, there is a lack of data in South Africa, which justifies continuous research, adherence to guidelines, and efforts to improve quality of care.[8]

CLASSIFICATION OF PERI-INFARCT ARRHYTHMIAS

Different types of arrhythmias can occur in the setting of STEMI. These can be categorised in the

following way.

Supraventricular tachyarrhythmias include sinus tachycardia, premature atrial contractions, atrial flutter, and atrial fibrillation.

Bradyarrhythmias include sinus bradycardia, junctional bradycardia, and atrioventricular blocks (AV).

Atrioventricular blocks are further subdivided into: first degree AV block, second degree AV block, and third-degree AV block.

Intraventricular conduction blocks are the left anterior fascicular block, left posterior fascicular block, right bundle branch block (RBBB), and left bundle branch block (LBBB).

Ventricular arrhythmias include premature ventricular contractions (PVCs), accelerated idioventricular

rhythm, ventricular tachycardia, and ventricular fibrillation.

SUPRAVENTRICULAR TACHYARRHYTHMIAS

Premature atrial contractions may occur before the development of paroxysmal supraventricular tachycardia, atrial flutter, or atrial fibrillation. In this setting, no specific therapy is indicated because they are usually benign. However, attention should be given to identify the underlying disease process, particularly occult heart failure.

Paroxysmal supraventricular tachycardia in the setting of AMI is uncommon except for atrial fibrillation and atrial flutter. Pharmacological therapy includes the use of adenosine when hypotension is not present to cardiovert the rhythm or slow down the heart rate to diagnose the rhythm. In patients with severe heart failure or hypotension, synchronised electrical cardioversion is required.[10,11]

Atrial flutter occurs in the setting of AMI. It is defined as an abnormal cardiac rhythm characterised by rapid, regular atrial depolarisation at a characteristic rate of approximately 300 beats/min and a regular ventricular rate of about 150 beats/min. It is usually transient and results from the sympathetic overstimulation of the atria. Although there is a lack of data on the incidence and prevalence of atrial flutter in STEMI, the treatment strategies for persistent atrial flutter are similar to atrial fibrillation.

Atrial fibrillation (AF) is a chaotic, irregular atrial rhythm at a rate of 300-600 beats per minute. The onset of atrial fibrillation in the first hours of AMI is usually caused by left ventricular (LV) failure, ischaemic injury to the atria, or right ventricular (RV) infarction. The presence of atrial fibrillation during AMI is associated with an increased risk of mortality with a crude mortality rate of about 60.7 per 1000 person-years.[12] It is also important to note that the risk of new-onset atrial fibrillation is also highest after two months of the AMI and decreases by the twelfth month[12] or stable patients with either AF or atrial flutter, controlling the ventricular rate is the immediate objective. Amiodarone or beta-blockers can be used to achieve ventricular rate control.[13,14]AF and atrial flutter also confer an increased risk ofthromboembolism. Therefore, anticoagulation therapy has to be started if not contraindicated.

BRADYARRHYTHMIAS

Sinus bradycardia is defined as a sinus rhythm with a resting heart rate of 60 beats per minute or less. However, few patients become symptomatic until their heart rate drops to less than 40 beats per minute. Historical data has shown sinus bradycardia to be associated with a prevalence of 15% - 25% percent of acute myocardial infarction cases.[15] It is a common arrhythmia in patients with inferior or posterior acute myocardial infarctions because, in approximately 60% of people, the right coronary supplies the SA node.[15] The highest incidence of sinus bradycardia is observed in the first 1-2 hours after AMI. In the early phases of an AMI, the resultant sinus bradycardia may be protective. The mechanism is probably due to cardiac vagal stimulation. When emergency treatment is required for symptomatic bradycardia, IVI atropine remains the first-line drug therapy, and transcutaneous or temporary pacing is usually indicated if the patient fails to respond to atropine.[16]

First degree AV blocks are characterised by prolongation of the PR interval to longer than 200 milliseconds. First-degree AV block at the level of the AV node is attributed to occlusion of the right coronary artery or the circumflex artery as these arteries give rise to the AV nodal artery. No specific therapy is indicated unless a hemodynamic compromise is present as the condition is usually transient.[16]

Second-degree heart block is characterised by the failure of some atrial impulse to reach the ventricles. It is divided into the following two subtypes, Mobitz type 1 and Mobitz type 2.

In Mobitz type 1 (Wenckebach AV block), there is PR interval prolongation on consecutive heartbeats, followed by a non-conducted P wave. It is usually associated with a narrow QRS complex attributable to a nodal block and is most commonly associated with an inferior MI. Wenckebach AV block does not necessarily require treatment if the heart rate is adequate for perfusion. If the heart rate is inadequate, the immediate recommended treatment is intravenous atropine. Transcutaneous or temporary transvenous pacing is rarely required as historical data demonstrates the condition to be benign.[16,17]In Mobitz type 2 AV block, the PR interval remains unchanged in relation to a non-conducted P wave, and a wide QRS complex usually characterises this. It is almost always associated with anterior infarction and has an incidence of about 5% in STEMI patients.[18] This type of block often progress suddenly to a complete heart block. Mobitz type 2 AV block is associated with a poor prognosis.

Therefore, hemodynamically unstable patients should be treated immediately with transcutaneous or temporary pacing and pharmacological therapy to support the cardiovascular status of the patient.

Third-degree AV block (a complete heart block) is characterised by no atrial impulse conduction to the ventricles. It occurs in about 3.8% of patients with inferior STEMI, and approximately 0.9% of patients with anterior STEMI.[19] A complete heart block in patients with an inferior MI is associated with narrow QRS compared to an anterior MI, which is associated with a wide QRS. Historically data demonstrates that a third-degree block with anterior STEMI is associated with high mortality both from pump failure and the arrhythmia itself.[19,20]

INTRAVENTRICULAR BLOCKS

Conduction from the His bundle is transmitted through 3 fascicles. The anterior division of the left bundle, the posterior division of the left bundle, and the right bundle. An abnormality of electrical conduction through any of these bundles causes a bundle branch block (BBB). A BBB may precede AMI. A BBB during AMI is associated with increased mortality with an overall mortality rate of 13.2% at 30 days.[21,22]

Isolated left anterior fascicular block (LAFB) may occur in patients with a significant lesion of the left anterior descending coronary artery (LAD).[23] This was noted in patients who underwent coronary angiography. It is also associated with an increased incidence of AF. Isolated left posterior fascicular block (LPFB) can occur in patients with AMI. The blood supply to the posterior fascicle is larger than that to the anterior fascicle. Therefore, the block is associated with a relatively large infarct and high mortality rates.[23,24]

The right bundle branch receives its dominant blood supply from the LAD artery. Therefore, a new right BBB (RBBB), seen in approximately 42% of patients with AMI, suggests a large infarct territory.[22,23,24] However, progression to complete AV block is uncommon. In patients who develop an anterior MI and a new RBBB, the substantial risk of death is mostly from cardiogenic shock, most likely due to the large size of the myocardial infarct.

The combination of RBBB and LAFB is known as a bi-fascicular block and is observed in approximately 5.5% of patients. A bi-fascicular block may occur with the occlusion of the proximal LAD coronary artery and is associated with a poor prognosis.[22,23,24] The risk of developing complete AV block is heightened, however complete block is still uncommon. Bifascicular block in the presence of a first-degree AV blockis called a trifascicular block. There is a lack of data regarding the incidence of a trifascular block.

VENTRICULAR ARRHYTHMIAS

Ventricular arrhythmias (VA) are abnormal rapid heart rhythms that originate in the ventricles. VA includes premature ventricular arrhythmias, accelerated idioventricular rhythm, ventricular tachycardia, and ventricular fibrillation. VA most commonly occurs in the early phase of ischaemia, when patients with acute MI are at the most significant mortality risk.[25,26] Early reperfusion therapy has significantly decreased the incidence of VA; however, 10% of post-MI survivors remain at risk of sudden cardiac death over the next months or years secondary to ventricular tachycardia (VT) or ventricular fibrillation (VF).[26] The incidence of ventricular arrhythmia is directly proportional to the infarct size and inversely related to the left ventricular ejection fraction (LVEF).[26]

Premature ventricular contractions (PVCs) were previously considered to represent an arrhythmic warning and impending malignant ventricular arrhythmias. However, these presumed "warning" arrhythmias are frequently observed in patients with AMI who never develop VF. VF often occurs without antecedent premature ventricular ectopy. Prophylactic suppression of PVCs with anti-arrhythmic drugs is no longer recommended as prophylaxis has been associated with an increased risk of fatal bradycardia or asystole because of the suppression of escape pacemakers.[27]

Given this evidence, most clinicians pursue a conservative course when PVCs are observed in a patient with AMI. Instead, attention should be directed at correcting any electrolyte or metabolic abnormalities, plus identifying and treating recurrent ischemia.

Accelerated idioventricular rhythm (AIVR) occurs in about 20% of patients who develop an AMI.[28] This pattern is defined by three or more ventricular complexes, with a wide, regular QRS complex of more than 120ms. The AIVR rate is higher than the intrinsic ventricular rate (40bpm). Hence it is accelerated; however, it remains less than 110 beats per minute.[28] Most AIVR episodes are short and terminate spontaneously. They occur in similar frequency in both anterior and inferior myocardial infarctions. The presence of an AIVR does not affect the patient's prognosis. Beta-blocker therapy can help reduce the AIVR burden, especially in patients with hemodynamic instability and persistent AIVR.[28] Administration of an anti-arrhythmic drug to treat AIVR can lead to hemodynamic deterioration and sinus bradycardia; therefore, the mainstay of treatment is to establish early reperfusion and correction of electrolyte abnormalities. This rhythm occurs more frequently in patients with early reperfusion, but it is neither specific nor sensitive as a marker for reperfusion. Temporary pacing is not recommended unless clinical hypotension ensues. Treatment with an anti-arrhythmic is also not recommended, as this can lead to significant bradycardia.[28]

Ventricular tachycardia (VT) is defined as three or more ventricular ectopic beats associated with a broad complex tachycardia with a rapid heart rate greater than 100 bpm and QRS interval greater than 120 milliseconds. Morphologically VT can be classified as monomorphic VT, characterised by a QRS morphology that is the same because there is a single ventricular focus verse a polymorphic VT in which there are multi-ventricular foci with the resultant QRS complex varying in amplitude, axis, and duration.

VT can be classified by duration. Non-sustained VT is defined as three or more ventricular ectopic beats at a rate greater than 100 bpm but lasting less than 30 seconds. Non sustained VT that occurs immediately after the peri-infarction period was independently associated with increased risk of death and other cardiovascular outcomes, including stroke.[27] Sustained VT is defined as three or more ventricular ectopic beats at a rate greater than 100bpm lasting more than 30 seconds. Sustained VT is associated with an in-hospital mortality rate of 20.3%.[25] Prescribing beta-blockers in the first 24 hours of AMI has been associated with a decrease in in-hospital mortality without worsening heart failure.

There is no added benefit with the use of amiodarone over and above concomitant beta-blocker therapy.[25,26] However, emergency treatment for sustained VT is mandatory because it can progress to ventricular fibrillation. Rapid polymorphic VT (rate>150) associated with hemodynamic instability should be treated with unsynchronised cardioversion at 200J. Monomorphic VT should be treated with synchronised cardioversion at 100J. If sustained VT is well-tolerated, amiodarone is the anti-arrhythmic drug of choice, and electrolyte/acid-base status and hypoxia should be corrected.

The rationale for the study

There is a lack of data from sub-Saharan Africa on the prevalence of arrhythmias in acute STEMI and the associated coronary artery territory. Data that is available is from developed countries.

In this study, the goal is to characterise the anatomical territory, e.g., anterior myocardial infarct/posterior myocardial infarct, and the arrhythmia associated with that territory in addition to assessing the outcome of the arrhythmia, that is, whether it resolved spontaneously; an anti-arrhythmic drug was required or whether pacing was required.

STUDY AIM

1. The study aims to describe the types of arrhythmic complications in acute STEMI at Charlotte Maxeke Johannesburg Academic Hospital.

OBJECTIVES

1. To describe the anatomical coronary artery occlusive lesions associated with the various arrhythmias identified in the peri-infarct period.
2. To determine the short-term mortality and morbidity (temporary or permanent pacemaker implantation) outcomes associated with the reported arrhythmias and the anatomical coronary artery occlusions, causing the acute STEMI.

RESEARCH METHOD

STUDY DESIGN

This research will be a retrospective cohort study.

STUDY SITE

Division of Cardiology, Charlotte Maxeke Johannesburg Academic Hospital (CMJAH), Johannesburg, South Africa.

STUDY POPULATION

All acute STEMI patients who presented at CMJAH who had baseline ECGs over five years from 1st January 2015 to 31st December 2019.

METHODOLOGY

Relevant data for patients using case files who meet the above inclusion criteria will be collected from the catheterisation unit in cardiology. A random case number of these patients will be used when structuring the database, so that patient confidentiality about their diagnosis is protected. Information will be recorded on the RedCap database (See attached RedCap Appendix).

Biochemistry results, specifically those that increase the risk of STEMI, for example, hypercholesterolemia, urea, and electrolytes, will be taken from the patients' files.

Inclusion criteria

All adult patients who are 18 years and older with a confirmed diagnosis of STEMI will be eligible to participate in the study.

Exclusion criteria

Patients with missing data, for example, those with absent ECG studies, will be excluded from the study.

STATISTICAL ANALYSIS

Means and standard deviations will be used for normally distributed data. Data will be expressed numerically and as percentages for straightforward interpretation. Medians with quartiles will be used on data that is non-normally distributed. Analysis of variance (ANOVA) and student's t-test will be used to test the intergroup proportion difference. Univariate and multivariate logistical regression will be used in order to determine clinical factors associated with arrhythmias and STEMI. A p-value of <0.05 will be considered statistically significant. Data collection will be done using RedCap software, and statistical analysis will be done using STATA version 16.

ETHICAL CONSIDERATION

Permission to conduct this study will be obtained from the HOD of cardiology, HREC, and the CEO of CMJAH

TIMELINE

2020	JUNE	JULY	AUG	SEP	OCT	NOV	DEC	JAN	FEB
LITERATURE REVIEW									
PREPARING PROTOCOL									
PROTOCOL ASSESSMENT									
ETHICS									
DATA COLLECTION									

DATA ANALYSIS									
WRITE UP									

FUNDING

The study is retrospective. It includes reviewing existing patient files. Therefore, the researcher does not anticipate any cost hence no need for additional funding.

REFERENCES

1. Mendis SPP, Norrving B. Global atlas on cardiovascular disease prevention and control. Geneva: World Health Organization, 2011.

2. Eckart RE, Shry EA, Burke AP, McNear JA, Appel DA, Castillo-Rojas LM, et al. Sudden death in young adults: an autopsy-based series of a population undergoing active surveillance. J Am Coll Cardiol. 2011;58(12):1254-61.

3. Brugada J, Katritsis DG, Arbelo E, Arribas F, Bax JJ, Blomström-Lundqvist C, et al. 2019 ESC Guidelines for the management of patients with supraventricular tachycardia. The task force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC). Eur Heart J. 2020;41(5):655-720.

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

5. Schamroth C. ACCESS South Africa investigators. 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.

6. 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.

7. Mandelzweig L, Battler A, Boyko V, Bueno H, Danchin N, Filippatos G, et al. The second Euro Heart Survey on acute coronary syndromes: Characteristics, treatment, and outcome of

patients with ACS in Europe and the Mediterranean Basin in 2004. Eur Heart J. 2006;27(19):2285-93.

8. Shavadia J, Yonga G, Otieno H. A prospective review of acute coronary syndromes in an urban hospital in sub-Saharan Africa. Cardiovasc J Afr. 2012;23(6):318-21.

9. Jernberg T, Johanson P, Held C, Svennblad B, Lindbäck J, Wallentin L, et al. Association between adoption of evidence-based treatment and survival for patients with ST-elevation myocardial infarction. JAMA. 2011;305(16):1677-84.

10. López-Sendón J, Swedberg K, McMurray J, Tamargo J, Maggioni AP, Dargie H, et al. Expert consensus document on beta-adrenergic receptor blockers. Eur Heart J. 2004;25(15):1341-62.

11. 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.

12. Benjamin EJ, Wolf PA, D'Agostino RB, Silbershatz H, Kannel WB, Levy D. Impact of atrial fibrillation on the risk of death: the Framingham Heart Study. Circulation. 1998;98(10):946-52.

13. January Craig T., L. Samuel Wann, Hugh Calkins, Lin Y. Chen et al:2019 AHA/ACC/HRS Focused guideline for the management of patients with atrial fibrillation. J Am Coll Cardiol 74(1):104–32.

14. Fuster Valentin, and Kay G. Neal. 2001. ACC/AHA/ESC Guidelines for the management of patients with atrial fibrillation: Developed in collaboration with the north American Society of Pacing and Electrophysiology. Circulation 104(17):2118–50.

15. Adgey AA, Geddes JS, Mulholland HC, Keegan DA, Pantridge JF. Incidence, significance, and management of early bradyarrhythmia complicating acute myocardial infarction. Lancet. 1968;2(7578):1097-101.

16. Kusumoto FM, Schoenfeld MH, Barrett C, Edgerton JR, Ellenbogen KA, Gold MR, et al. 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay: A report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines and the Heart Rhythm Society. Circulation. 2019;140(8):e382-e482.

17. Feigl D, Ashkenazy J, Kishon Y. Early and late atrioventricular block in acute inferior myocardial infarction. J Am Coll Cardiol. 1984;4(1):35-8.

18. Singh SM, FitzGerald G, Yan AT, Brieger D, Fox KA, López-Sendón J, et al. High-grade atrioventricular block in acute coronary syndromes: insights from the Global Registry of Acute Coronary Events. Eur Heart J. 2015;36(16):976-83.

19. Harikrishnan P, Gupta T, Palaniswamy C, Kolte D, Khera S, Mujib M, et al. Complete heart block complicating ST-segment elevation myocardial infarction: Temporal trends and association with in-hospital outcomes. JACC Clin Electrophysiol. 2015;1(6):529-38.

20. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheinman MM, DeSanctis RW, et al. The clinical significance of bundle branch block complicating acute myocardial infarction. 2. Indications for temporary and permanent pacemaker insertion. Circulation. 1978;58(4):689-99.

21. Xiang L, Zhong A, You T, Chen J, Xu W, Shi M. Prognostic significance of right bundle branch block for patients with acute myocardial infarction: A systematic review and meta-analysis. Med Sci Monit. 2016;22:998-1004.

22. Melgarejo A, Galcerá J, Consuegra L, Nuria A, Ángela D, Germán E, et al. Relation of new permanent right or left bundle branch block on short- and long-term mortality in acute myocardial infarction bundle branch block and myocardial infarction. Am J Cardiol. 2015; 116(7):1003-9.

23. Lévy, S. 'Bundle branch blocks and/or hemiblocks complicating acute myocardial ischemia or infarction'. 2018 J Interv Card Electrophysiol (52):287–292.

24. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheinman MM, DeSanctis RW, et al. The clinical significance of bundle branch block complicating acute myocardial infarction. 1. Clinical characteristics, hospital mortality, and one-year follow-up. Circulation. 1978;58(4):679-88.

25. Santangeli P, Rame JE, Birati EY, Marchlinski FE. Management of ventricular arrhythmias in patients with advanced heart failure. J Am Coll Cardiol. 2017;69(14):1842-60.

26. Bhar-Amato, Justine, William Davies, and Sharad Agarwal. 2017. Ventricular arrhythmia after acute myocardial infarction: The perfect storm. Arrhythmia & Electrophysiology Review 6(3):134–39.

27. Al-Khatib, Sana M., William G. Stevenson, Michael J. Ackerman, William J. Bryant, David J. Callans, Anne B. Curtis, Barbara J. Deal, Timm Dickfeld, Michael E. Field, Gregg C. Fonarow. 2017 AHA/ACC/HRS Guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. J Am Coll Cardiol 72(14):91–220.

28. Chen M, Gu K, Yang B, Chen H, Ju W, Zhang F, et al. Idiopathic accelerated idioventricular rhythm or ventricular tachycardia originating from the right bundle branch: unusual type of ventricular arrhythmia. Circ Arrhythm Electrophysiol. 2014;7(6):1159-67.