UNIVERSITI TEKNOLOGI MARA

UNIVERSITI TEKNOLOGI MARA

SMALL DENSE LOW DENSITY LIPOPROTEIN PROFILE IN YOUNG ADULTS WITH ACUTE CORONARY SYNDROME: A CASE CONTROL STUDY

DR MOHD YAZID BIN ZAMRI

2014947265

MASTERS IN INTERNAL MEDICINE

MD 771

OCTOBER 2020

ABSTRACT

Background and Aims. Small dense LDL (sdLDL) has grown in consensus to be a more specific atherogenic subclass of low density lipoprotein (LDL-C) involved in the pathogenesis of atherosclerosis and its complication such as acute coronary syndrome. Measurement of sdLDL could potentially improve the management of risk in the primary and secondary prevention population. This study aims to describe the sdLDL level in young adults diagnosed with acute coronary syndrome (ACS). and its relationship with inflammation in young ACS.

Methodology. This was a single-centered, matched risk case–control, cross-sectional study involving 100 young adults (age ≤50 years) of whom 50 were diagnosed with ACS and 50 without ACS. Direct LDL-c, lipid profile and a biomarker of inflammation, high sensitive C-Reactive Protein (hsCRP) were measured by an automated analyzer (c501, Roche Diagnostics, Germany) and sdLDL level was calculated (calc-sdLDL) using a validated formula.

Results: Calculated sdLDL level (represented in median and IQR) was significantly higher among subjects with ACS in comparison to those without (0.97 (0.69- 1.29) vs 0.86 (0.62-1.04), p=0.03). There were no significant difference in the calculated LDL and direct LDL-C between both groups. HsCRP (represented in median and IQR) were higher among those with ACS compared to those without [(45.07 (4.89 -76.08) vs 2.165 (1.16-5.35), p=<0.001). There was no association between hsCRP and calc-sdLDL in both groups, whereby in the ACS group, the correlation coefficient (r_s) is 0.15, with p-

ACKNOWLEDGEMENT

Praises to God for the blessing and opportunity to complete my Master in Internal Medicine. I am very thankful to my clinical supervisor, Prof. Dr. Sazzli Shahlan Kasim for contributing his knowledge and ideas in constructing this research. I would also like to express my gratitude to my co-supervisor, Associate Prof. Dr. Thuhairah Hasrah Abd Rahman for her relentless guidance through this research period. Not to be forgotten, Dr. Rodi Isa for providing his brilliant ideas and input in constructing the methodology of this research.

I would like to thank physicians for their guidance and assistance. My appreciation also goes out to for helping to ease my research flow. I am very grateful to have worked in Hospital Sungai Buloh.

Lastly, to my wife, thank you for your support and understanding through this Master program. To my family and friends, your support is priceless, and I will cherish it forever.

TABLE OF CONTENT

CHAPTER ONE

INTRODUCTION

1 Background of the study

Coronary artery disease (CAD) remains the principle cause of death in developed and weloping countries. Atherosclerosis is a well-known pathophysiology of CAD described as aque formation composed by lipid core and fibrous cap. Specifically, low density lipoprotein lolesterol (LDL-C) is the primary contributor of the lipid component in the pathogenesis of herosclerosis. There is recent understanding regarding the influence of LDL size on herogenesis, suggesting that small dense LDLs are associated with atherogenicity and a better omarker as Acute Coronary Syndrome (ACS) predictor h

Acute Coronary syndrome (ACS) is a spectrum of myocardial ischemia depending on the verity of coronary artery occlusion. ACS can range from unstable angina (UA), while myocardial farction (MI) can vary from Non ST-elevation MI (NSTEMI) and ST- elevation MI (STEMI). alike MI where myocardial injury is evident, unstable angina does not cause the release and rise 'cardiac biomarkers. MI is a diagnosis made clinically on the basis of electrocardiograph (ECG) idings, cardiac biomarkers and evidence of intracoronary' thrombus found during autopsy in the se of mortality [2>3].

For the entire spectrum of ACS, clinical history is the single-most important diagnostic mponent. Angina pain is the prerequisite to ECG findings and cardiac biomarkers when agnosing ACS. However, as patients may present atypically, other angina-equivalent complaints ould be sought including shortness of breath, palpitations and syncope/pre-syncope. ECG anges in UA and NSTEMI include deep symmetrical T inversions, dynamic ST changes and ST pressions of >0.5mm in two or more contiguous leads. Other suggestive ECG changes include w onset bundle branch block and cardiac arrhythmias. For STEMI, the hallmark ECG changes 2 ST elevation and/or presumed left bundle branch block (LBBB).