



The Atherogenic Index of Plasma (AIP), Visceral Adiposity Index (VAI), Lipid Accumulation Product (LAP) for assessment of Cardiovascular risk: A Comparative Analysis between Normal Subjects and Patients with Acute Myocardial Infarction

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ABSTRACT

Acute Myocardial Infarction (AMI) is caused by reduced blood flow in a coronary artery and it has been the leading cause for morbidity and mortality worldwide for the last 20 years. Aim of this study was to compare the Atherogenic Index of Plasma (AIP), Visceral Adiposity Index (VAI), Lipid Accumulation Product (LAP) and lipid profile parameters between normal subjects and patients with AMI admitted to a tertiary care hospital in Sri Lanka and to find the correlations of AIP, VAI, LAP with lipid parameters. A cross-sectional study was conducted involving a total of 100 patients with AMI and 100 age and gender matched healthy subjects of age between 18 and 85 years. Blood samples were drawn from patients with AMI admitted to Colombo South Teaching Hospital within 24 hours of the onset of chest pain. The total cholesterol (TC), triglycerides, LDL, and HDL were analyzed and used with BMI and waist circumference (WC) for calculating VAI, AIP and LAP. The HDL level of cases was significantly lower when compared to HDL level of control subjects ($p < 0.05$). Significant differences were not observed in TC and LDL between cases and controls. The significant differences in Triglycerides, HDL, AIP, VAI and LAP were observed only between female patients with AMI and female control subjects ($p < 0.05$). Mean AIP, VAI and LAP of cases showed significant correlations only with triglyceride and HDL. The AIP, VAI, and LAP can be used for the assessment of cardiovascular risk in patients, especially in female subjects.

KEYWORDS: *Atherogenic Index of Plasma (AIP), Visceral Adiposity Index (VAI), Lipid Accumulation Product (LAP), Acute myocardial infarction, Lipid profile*

1 INTRODUCTION

1.1 Prevalence of Cardiovascular diseases

An acute myocardial infarction (AMI) is a condition caused by reduced blood flow in a coronary artery, usually due to atherosclerosis, thrombus, or embolus formation. AMI is irreversible damage of myocardial tissue caused by prolonged ischemia and hypoxia (Palasubramaniam, Wang and Peter 2019). Worldwide, cardiovascular diseases (CVD) are the main cause of morbidity and mortality, accounting for nearly one-third of all fatalities. Globally, cardiovascular-related deaths have steadily increased by more than one-third from 1990 to 2019 (Minja et al. 2022). It is estimated that 17.9 million people died from cardiovascular diseases in 2019, representing 32% of all global deaths (Ramesh and Kosalram 2023). Among a cohort of 4,890 patients with acute myocardial infarction (AMI) studied by Cai et al., using the MIMIC III and IV databases, the 1-year all-cause mortality rate was 33.2% in women compared to 22.5% in men. After adjusting for confounders, men had about an 18% lower risk of death than women during this period, indicating significantly higher mortality among women (Cai et al 2025). Sri Lanka has one of the highest rates of cardiovascular disease mortality among all countries. Ischemic heart disease (IHD) is the leading cause of death in Sri Lanka, accounting for 27.6 deaths per 100,000 population (Medagama et al. 2015).

1.2 The importance of exploring anthropometric indices as predictors of acute myocardial infarction risk

Atherosclerosis stands out as a primary cause of AMI (Van der Schoot, Anthonio and Jessurun, 2020). Dyslipidemia is the major risk factor for various cardiovascular diseases, and it is a powerful predictor of cardiovascular outcomes following an AMI. High triglyceride, TC, LDL-C, and low HDL-C levels can contribute to the formation of atherosclerotic plaques (Zhong et al. 2017). However, lipid profile parameters do not provide adequate and reliable information about the state of atherosclerosis because there may be variations due to various reasons such as diet, medical conditions and other causes such as genetics, smoking, etc. (Rafieian-Kopaei et al. 2014). Patients with AMI also may not always have high triglyceride, TC, LDL-C, and low HDL-C levels. Therefore, additional methods beyond the conventional lipid profile are needed to assess AMI risk. Instead of solely relying on lipid profile parameters, it is crucial to consider additional indices such as the Visceral Adiposity Index (VAI), Lipid Accumulation Product (LAP), and Atherogenic Index of Plasma (AIP), which are derived from both lipid profile and anthropometric data, for assessing acute myocardial infarction risk. This is especially important because research has revealed that VAI demonstrates a robust independent association with cardiovascular risk (Zhang et al. 2022). Clinical studies have also reported a link between visceral adipose tissue and cardiac structure and function (Amato and Giordano 2014). VAI can be considered as a reliable marker for predicting CVD (Cai et al. 2022). The Atherogenic Index of Plasma (AIP) serves as a robust and independent predictor for

coronary artery diseases, making it a valuable marker of plasma atherogenicity (Cai et al. 2019, Fu et al. 2021). Lipid Accumulation Product (LAP), emerging as a novel index for predicting Cardiovascular Diseases (CVD), has garnered significant interest among researchers (Shi et al. 2022). Some studies evaluate LAP as a predictor of cardiovascular outcomes and mortality (Papathanasiou et al. 2024).

The aim of this study was to find whether LAP, VAI and AIP can be used for assessment of cardiovascular disease risk by comparing these indexes in selected patients with AMI with these indexes in healthy individuals. If these indexes showed considerable alternation between patients with AMI and healthy individuals, those indexes can be used as more effective biomarkers to predict CVD. No previous studies have compared all three indices—Visceral Adiposity Index (VAI), Lipid Accumulation Product (LAP), and Atherogenic Index of Plasma (AIP)—together in patients with acute myocardial infarction (AMI) in Sri Lanka, and conducting such research could help identify individuals most vulnerable to cardiovascular diseases (CVD), ensuring they receive appropriate treatment and thereby reducing premature deaths.

2 MATERIALS AND METHODS

2.1 Subjects

A cross-sectional study was carried out involving patients with AMI admitted to Colombo South Teaching Hospital (CSTH) from June to August 2022 and normal subjects who were recruited through home visits

conducted in the community surrounding the University of Sri Jayewardenepura. The total of 100 patients with AMI and 100 age and gender-matched healthy subjects were recruited for the study after obtaining their written informed consent. The study protocol (MLS/3/2022) was approved by Ethics Review Committee, Faculty of Medical Sciences, University of Sri Jayewardenepura, Sri Lanka. The sample analysis was carried out at the Department of Medical Laboratory Sciences, Faculty of Allied Health Sciences, University of Sri Jayewardenepura, Sri Lanka. Patients younger than 18 years and pregnant women were excluded from the study. BMI values of all participants were calculated after measuring their weight and height.

2.2 Measurements

In accordance with WHO guidelines, anthropometric measurements were obtained using calibrated equipment. BMI was calculated from height and weight. A flexible plastic tape was used to measure the WC to the nearest 0.1 cm, midway between the iliac crest and the lower rib margin.

Blood samples were collected from patients with AMI and healthy control subjects under aseptic conditions to assess lipid profile parameters and VAI, AIP and LAP were calculated using BMI and WC. The following formulas were used to calculate VAI, AIP and LAP (Dieny, Tsani and Suryawa 2022, Niroumand et al. 2015).

$$\text{Females; } VAI = \left\{ \frac{WC}{36.58 + (1.89 \times BMI)} \right\} \times \left\{ \frac{TG}{0.81} \right\} \times \left\{ \frac{1.52}{HDL} \right\}$$

$$\text{Males; } VAI = \left\{ \frac{WC}{36.68 + (1.88 \times BMI)} \right\} \times \left\{ \frac{TG}{1.03} \right\} \times \left\{ \frac{1.31}{HDL} \right\}$$

Male LAP = [waist (cm)–65] × TG concentration (mmol/l)

Female LAP=[waist(cm)–58] × TG concentration (mmol/l)

AIP = log (TG/HDL)

Reference range for VAI

Males: ≤ 2.52

Females: ≤ 2.52

(There is no specialized unit for VAI)

(Amato et al. 2010)

Reference range for LAP

Males: 20.10 to 63.89 cm·mmol/L

Females: 25.16 to 31.59 cm·mmol/L

(Kaneva and Bojko 2021)

Reference range for AIP

Males/females: ≤ 0.11

(There is no specialized unit for AIP)

(ODX Research 2025)

The serum was aliquoted and stored in -80°C freezer until analysis. The lipid profile samples were analyzed within two-days of collection using the KONELAB 20XT analyzer, which included quality controls and calibrators to ensure accuracy (KONELAB-20XT BioLabo SAS, France).

2.3 Statistical analysis

Statistical analysis was carried out using SPSS version 26.0 (SPSS Inc., Chicago, IL, USA). The distribution of variables was assessed using the Shapiro–Wilk test to determine normality. For variables that were normally distributed, results were expressed as mean $\pm$ standard deviation, and independent samples t-tests were used for group comparisons. Pearson correlation was used to assess the association between AIP, VAI, LAP and lipid profile

parameters. A p-value less than 0.05 was deemed statistically significant in the analysis.

3 RESULTS & DISCUSSION

Of the 200 participants, 112 were females and 88 were males. The mean age of the normal control subjects and patients with AMI were 63.72 ± 11.98 and 64.84 ± 13.04 respectively. The cases and controls did not differ significantly on BMI ($p=0.055$). Mean BMI for normal control and AMI cases were 24.78 ± 4.85 (kg/m^2) and 24.16 ± 5.12 (kg/m^2) respectively.

The mean values of biochemical parameters in the two groups are described in Table 1.

As shown in Table 1, no significant differences were observed in total cholesterol and LDL levels between patients with acute myocardial infarction (AMI) and control subjects. These findings differ from several previous studies which reported significantly higher total cholesterol and LDL levels in AMI patients compared to controls (Kheraj Mal et al. 2019, Wang et al. 2020).

In our study, triglyceride levels were significantly higher and HDL levels were significantly lower in patients with AMI, particularly among females. These observations align with previous research indicating that elevated triglycerides and reduced HDL cholesterol are important risk factors for coronary artery disease, especially in women (Wang et al. 2020, Shirafkan, Marjani and Zaker 2012).

Table 1. Comparison of lipid profile parameters of males and females between normal control group and patients with acute myocardial infarction (AMI)

	Normal Controls Mean $\pm$ SD (n=100)	AMI cases Mean $\pm$ SD (n=100)	p value
Total cholesterol (mg/dL)			
Female	162.09 $\pm$ 18.82	153.86 $\pm$ 56.77	0.509
Male	148.41 $\pm$ 23.32	152.00 $\pm$ 41.58	0.752
Total subjects	155.63 $\pm$ 21.79	153.51 $\pm$ 50.58	0.803
Triglycerides (mg/dL)			
Female	96.56 $\pm$ 25.99	131.00 $\pm$ 41.17	0.001*
Male	110.12 $\pm$ 28.58	124.63 $\pm$ 51.56	0.304
Total subjects	101.49 $\pm$ 27.78	129.43 $\pm$ 45.27	0.001*
HDL (mg/dL)			
Female	52.48 $\pm$ 11.56	37.71 $\pm$ 11.85	0.001*
Male	47.00 $\pm$ 8.31	43.32 $\pm$ 13.71	0.335
Total subjects	50.41 $\pm$ 10.55	39.75 $\pm$ 12.65	0.001*
LDL (mg/dL)			
Female	90.22 $\pm$ 14.53	89.94 $\pm$ 49.90	0.979
Male	79.34 $\pm$ 20.55	83.76 $\pm$ 37.22	0.663
Total subjects	84.86 $\pm$ 18.33	87.87 $\pm$ 44.67	0.687

*P<0.05

High triglyceride levels contribute to plaque formation while low HDL impairs reverse cholesterol transport, increasing susceptibility to cardiovascular events. However, the literature is not entirely consistent; some studies have reported no significant difference in triglyceride levels between AMI patients and controls (Wang et al. 2020), suggesting that these lipid abnormalities may vary depending on the demographic and lifestyle factors. Overall, these findings highlight the complex relationship between lipid profiles and AMI risk and highlight the need for population-specific studies to guide cardiovascular risk assessment and management.

In this study, total cholesterol and LDL levels did not differ significantly between AMI

patients and controls, even though triglycerides were higher and HDL was lower. One reason could be population differences, such as genetics, diet, and lifestyle, which can affect baseline lipid levels. Another reason may be the use of lipid-lowering medications, which can reduce total cholesterol and LDL. The timing of blood sample collection might also matter. Finally, factors like body fat, physical activity, and metabolism may affect triglycerides and HDL more than total cholesterol and LDL.

The present study demonstrated that the Atherogenic Index of Plasma (AIP) was significantly elevated in patients with acute myocardial infarction (AMI) compared to controls, highlighting its potential role as a predictive marker for cardiovascular risk. These findings are consistent with previous research, where elevated AIP levels were associated with both the occurrence and severity of coronary artery disease (Ghugre and Zin 2012, Wang et al. 2020). Interestingly, the increase in AIP was particularly notable among female participants, suggesting gender-specific differences in lipid-related cardiovascular risk. While studies in elderly and middle-aged men have also identified AIP as a cardiovascular risk factor, some have reported its predictive power to be comparable or even inferior to conventional lipid parameters such as LDL cholesterol (Cai et al. 2019, Hong et al. 2022). This variability underscores the importance of considering demographic and population-specific factors when interpreting AIP values.

Table 2. Comparison of Atherogenic Index of Plasma (AIP), Visceral Adiposity Index (VAI) and Lipid Accumulation Product (LAP) values of males and females between control group and patients with acute myocardial infarction (AMI)

Index	Normal Control Mean $\pm$ SD (n=100)	AMI cases Mean $\pm$ SD (n=100)	p value
AIP			
Female	-0.10 $\pm$ 0.16	0.18 $\pm$ 0.23	0.001*
Male	0.00 $\pm$ 0.16	0.07 $\pm$ 0.27	0.265
Total subjects	-0.06 $\pm$ 0.16	0.15 $\pm$ 0.24	0.001*
VAI			
Female	1.59 $\pm$ 0.79	3.41 $\pm$ 1.65	0.001*
Male	1.38 $\pm$ 0.54	1.90 $\pm$ 1.33	0.136
Total subjects	1.49 $\pm$ 0.69	2.78 $\pm$ 1.68	0.001*
LAP			
Female	27.40 $\pm$ 12.30	44.89 $\pm$ 21.40	0.001*
Male	25.69 $\pm$ 11.33	25.36 $\pm$ 17.63	0.947
Total subjects	26.55 $\pm$ 11.65	36.59 $\pm$ 22.07	0.010*

*P<0.05

Similarly, the Visceral Adiposity Index (VAI) was significantly higher among female AMI patients compared to controls, whereas male participants did not show a significant difference. This pattern aligns with prior observations that visceral fat accumulation may exert a stronger influence on cardiometabolic risk in women (Wang et al. 2022, Ho et al. 2016). Visceral adiposity has been consistently associated with insulin resistance, dyslipidemia, and inflammatory states, all of which contribute to cardiovascular events, making VAI a valuable gender-sensitive biomarker (Neeland, Poirier and Després 2019, Amato et al. 2011).

Lipid Accumulation Product (LAP) values exhibited a similar trend, with significantly higher levels observed among female AMI

patients, while males did not demonstrate a significant difference. Previous studies have reported comparable findings, emphasizing the stronger association between LAP and cardiometabolic outcomes in women, particularly postmenopausal females (Wehr et al. 2011, Bozorgmanesh, Hadaegh and Azizi 2010). The sex-specific differences in LAP may be explained by variations in fat distribution and storage capacity. Before menopause, women tend to store lipids in lower-body adipose tissue, which provides a protective buffer against ectopic lipid deposition. As LAP increases, surplus lipids may accumulate in non-adipose tissues, including the liver, heart, and vasculature, potentially disrupting cellular metabolism and contributing to cardiovascular risk (Papathanasiou et al. 2024).

Correlations of Atherogenic Index of Plasma (AIP), Visceral Adiposity Index (VAI), Lipid Accumulation Product (LAP) with lipid profile parameters are shown in Figures 1,2 and 3.

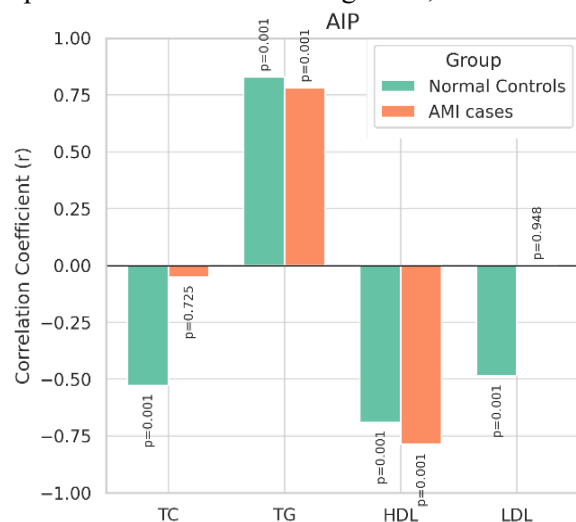


Figure 1. Correlations of Atherogenic Index of Plasma (AIP) with lipid profile parameters – Normal control vs AMI cases

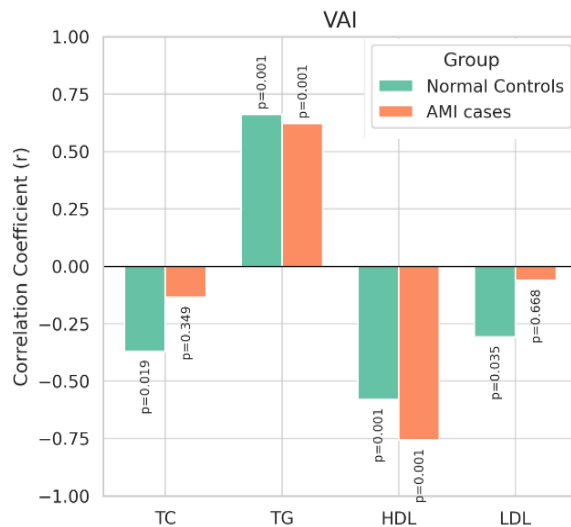


Figure 2. Correlations of Visceral Adiposity Index (VAI) with lipid profile parameters – Normal control vs AMI cases

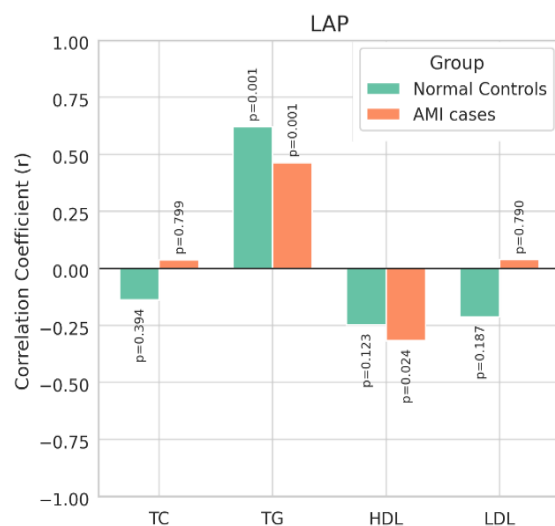


Figure 3. Correlations of Lipid Accumulation Product (LAP) with lipid profile parameters – Normal control vs AMI cases

In our study, the Atherogenic Index of Plasma (AIP) among patients with acute myocardial infarction (AMI) exhibited a positive correlation with triglycerides and a negative correlation with high-density lipoprotein (HDL) levels (Figure 1). A study done by Gaojun Cai et al., suggested a significant positive correlation of AIP with TC, triglyceride, LDL, and negative correlation

with HDL (Cai et al. 2017). Dabla et al. in 2021 reported that while the precise mechanism linking elevated AIP to heightened cardiovascular mortality risk remains unclear, potential explanations could involve its association with LDL-C particle size, insulin resistance, and metabolic syndrome. (Dabla et al. 2021).

According to our results, the mean VAI of control subjects showed significant correlations with lipid profile parameters (TC, triglyceride, HDL) except LDL, while VAI of patients with AMI showed significant correlations only with triglyceride and HDL (Figure 2). Klisic et al. conducted a study aiming to ascertain the link between an adverse lipid profile and glycemic control in individuals with type 2 diabetes mellitus. They reported that Visceral Adiposity Index (VAI) exhibited correlations with all lipid parameters. (Klisic et al. 2017). The differences in their findings when compared to the present study's, could be explained in relation to their use of diabetic patients.

In the current study, the mean Lipid Accumulation Product (LAP) of patients with acute myocardial infarction (AMI) showed a significant positive correlation with triglyceride levels and a significant negative correlation with HDL cholesterol (Figure 3). This aligns partially with the findings of Anik Ilhan, Yildizhan and Pekin (2018), who reported that LAP and Visceral Adiposity Index (VAI) were positively correlated with each other as well as with triglyceride and total cholesterol levels in lean women with polycystic ovary syndrome

(Anik Ilhan, Yildizhan and Pekin 2018). Our findings differ from that study, as we observed a positive correlation only between LAP and triglycerides, whereas no such correlation was found with HDL. Though there are no studies conducted specifically in AMI patients, Fonseka, Wickramasinghe and Dissanayake (2019) reported that the Visceral Adiposity Index (VAI) was significantly associated with lipid profile parameters in Sri Lankan women with polycystic ovary syndrome, suggesting its potential utility as a marker for lipid abnormalities (Fonseka, Wickramasinghe and Dissanayake 2019).

One limitation of the present study was the age imbalance within the study population, with over 80% of participants were aged 40 years and older. In addition, it did not include other myocardial infarction risk factors, such as diabetes, high blood pressure, smoking, or family history. These factors could have influenced the lipid profile parameters, AIP, LAP and VAI results. Not including those risk factors limits the ability of interpreting the findings. Future studies should consider these risk factors for a clearer assessment of myocardial infarction risk.

4 CONCLUSION & RECOMMENDATIONS

In this study, female patients with acute myocardial infarction (AMI) had higher triglycerides, lower HDL, and elevated Atherogenic Index of Plasma (AIP), Visceral Adiposity Index (VAI), and Lipid Accumulation Product (LAP) compared to

female controls. These findings indicate that these indices capture lipid profile parameters, AIP, LAP and VAI differences associated with AMI, especially in women. Male patients did not show significant differences in VAI or LAP, suggesting that these indices may reflect sex-specific metabolic changes during acute myocardial infarction.

It is important to note that this study only assessed patients during acute MI and did not evaluate other cardiovascular conditions, such as stroke, transient ischemic attack, or stable angina. Therefore, while elevated levels of AIP, VAI, and LAP indicate metabolic and lipid disturbances in women with AMI, these results cannot be directly interpreted as indicators of overall cardiovascular risk. Longitudinal studies following patients over time would be needed to determine whether these indices can reliably predict future myocardial infarction risk.

Despite this limitation, AIP, VAI, and LAP may serve as useful supplementary measures to traditional lipid profiles, particularly for identifying gender-specific patterns in metabolic risk during acute myocardial events. These indices could help clinicians better characterize female patients who experience AMI and guide further monitoring or preventive strategies. Future research with larger, balanced cohorts of males and females is recommended to validate these findings and clarify the clinical utility of these indices in long-term cardiovascular risk assessment.

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