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Original Research Article

A study of atherogenic index of plasma in myocardial infarction patients admitted in Silchar medical college, Assam, India

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ABSTRACT

Background: It is an established fact that dyslipidemia is one of the major risk factors for cardiovascular diseases like myocardial infarction. Major well-known alterations in lipid profile include high serum cholesterol, low density lipoprotein cholesterol (LDL-C), triglyceride and low high density lipoprotein cholesterol (HDL-C). Recently, it has been found that atherogenic index of plasma (AIP); which is a logarithmically transformed ratio of triglyceride and HDL-C; can predict cardio-vascular disease risk and can also be used for cardio-vascular disease risk stratification. In this study we have calculated the AIP from fasting lipid profile of patients suffering from myocardial infarction and tried to assess the correlation between AIP and myocardial infarction.
Methods: The study comprised of fifty patients suffering from myocardial infarction aged more than 18 with no prior history of cardiovascular diseases. We measured fasting lipid profile using Vitros 5600 full auto analyzer and calculated AIP using online AIP calculator. Statistical analysis was done using "Microsoft excel 2019" with add on. A significant percentage (66%) of myocardial infarction patients had higher AIP (>0.21) and fell in the high-risk group. We also got a significant relationship between AIP-triglyceride and AIP-HDL-C ($p<0.05$ considered as statistically significant).
Conclusions: AIP is high in myocardial infarction patients. In this group, besides high AIP, they also have low HDL-C and high triglycerides which are significant. So, AIP can be used as a tool for cardio-vascular disease risk stratification.

Keywords: AIP, Myocardial infarction, Triglyceride, High density lipoprotein

INTRODUCTION

The role of dietary fat in human body was described in ancient Indian literature "Bhagavad Gita" in 3500 BC.[1] Civilization has crossed a long way after that. Pure cholesterol was first isolated from a gall stone in 1784 by Francois Pelletier who was a French physician and chemist. At least 13 scientists received Nobel prize who were involved in the research work on cholesterol. The relation between cholesterol and atherosclerosis was first assumed by a scientist named Windaus in 1910. First genetic connection between cholesterol and myocardial infarction was discovered in 1939 by Carl Muller, a clinician from Norway. In the early part of 1950, a scientist of university of California, John Gofman discovered that not only elevated serum cholesterol level was responsible for heart attack, but raised serum LDL also causes heart attack and increased level of serum HDL lessens the chance of it.[1] With this the coronary event-cholesterol relationship was better understood.

The concept of risk factors in regards to cardiovascular disease was revealed in the "Framingham heart study" which is also considered as the mother of evidence-based medicine. This study shows the evidence that, a person with baseline high serum cholesterol level was more prone for myocardial infarction than those who had normal baseline serum cholesterol level.[2] Now in this

modern era of science it is now well established that atherogenic dyslipidemia is the main risk factor for coronary artery disease as atherogenic plaque causes stenosis of coronary artery and subsequently causes myocardial infarction. Well studied lipid profile alterations that may be responsible for coronary artery disease are high serum cholesterol and LDL, and low serum HDL. The role of serum triglyceride has not studied extensively compared to the other major parameters of lipid profile. Evidence suggests that high serum triglyceride level is associated with increased circulatory LDL and subsequently increase rate of cardiovascular events.[3]

AIP is logarithmically transformed ratio of triglyceride and HDL and is considered as a marker of atherogenicity. It reflects the degree of atherogenicity.[4] Small and dense LDL particle (sd-LDL) is proatherogenic as it is more susceptible for oxidation and subsequently with increase atherogenic ability.[5]

Increase in the concentration of small dense LDL level bears the CHD risk of 3-7 times regardless of the LDL cholesterol level in blood. Kwon et al established the relationship of small LDL with severity of coronary heart disease.[5] Atherogenic index of plasma (AIP) correlates well with the LDL particle size, so can be an indicator of small dense LDL which is responsible for atherogenic plaque formation.[6] AIP also determines the fractionated esterification rate of HDL.[7] Measurement of small dense LDL in the blood is a complicated procedure as well as an expensive one. So, by simply calculating AIP from lipid profile one can easily predict the risk for coronary artery disease. So, we can say that AIP can be more useful than lipid profile alone for risk stratification of myocardial infarction.

Among the non-communicable diseases, cardio vascular diseases (CVD) are one of the leading causes of death globally as well as in India. According to WHO data it takes near about 17.9 million lives globally.[8] Four out of five CVD deaths are due to myocardial infarction and stroke. Whereas globally age-standard death rate due to CVD is 235 per one lakh population, in India this is 272 per one lakh population which is much higher than the global rate. The prevalence of cardiovascular death is increasing in developing countries like India. In western countries 23% lost their lives before there 70th birthday due to cardio vascular disease, in India this percentage is 52%. As the treatment of cardiovascular diseases like myocardial infarction is expensive in countries like India, so prevention is the only effective way to minimize the causality. An effective way of prevention can increase country's productivity also. It is a well-established fact that atherosclerotic plaque is an important cause of cardiovascular diseases like myocardial infarction. So, AIP calculation can be an important and vital tool for risk stratification as it is directly proportional to the atherogenicity. It can be more sensitive than measuring and interpreting lipid profile alterations alone.[10,11] AIP is logarithmically transformed ratio of triglyceride and HDL which reflects the small dense LDL which is mainly responsible for atherosclerosis.

In this study we measured AIP from fasting lipid profile of fifty myocardial infarction patients admitted in Silchar medical college between March 2020 to August 2020. We aimed to see the percentage of myocardial infarction patients having AIP of high risk, intermediate risk and low risk group and also to find out any gender wise variation of AIP in those patients as well as to see the relation of severity of myocardial infarction with AIP in the terms of value of troponin I.

METHODS

This study was conducted in the department of biochemistry, Silchar medical college, Cachar, Assam. Fifty patients of MI were included in the study who were admitted in the medicine ward or ICU between March 2020 to August 2020 and fulfilled the inclusion criteria for the study as mentioned below.

Inclusion criteria

Patients aged more than 18 years suffering from myocardial infarction (documented cardiac troponin I positive patients) were included in the study.

Exclusion criteria

Patients with history of previous myocardial infarction and patients with history of any other cardiovascular diseases were excluded from the study.

Collection of samples

Fasting blood sample for serum lipid profile, creatinine and glucose was obtained from the patients in clot activator vial and fluoride vial respectively from antecubital vein under full aseptic precautions.

Analysis of samples

The samples were analyzed in Vitros 5600 full auto analyzer. In fasting lipid profile, serum cholesterol was measured using cholesterol oxidase, esterase, peroxidase method, triglyceride (TG) measured by colorimetric method, direct HDL was measured using colorimetric, PTA/$MgCl_2$ method. Cardiac troponin I was measured by immunometric method in analyzer whereas serum creatinine was measured using two-point rate-enzymatic method and blood glucose measured by glucose oxidase method in the said analyzer. All the tests were done after ensuring proper control for each test.

Atherogenic index of plasma was calculated based on the formula: AIP=log (TG/HDL-C)[3] Here we used online AIP calculator. AIP<0.11 considered as low risk, between 0.11 to 0.21 considered as intermediate risk, value>0.21

considered as high risk. A questionnaire was developed where relevant history of the patient was taken along with demographic profile. Informed written consent was also obtained from the patient or from the next of kin.

Statistical analysis

Statistical analysis was done using Microsoft excel 2019 with add ins. Continuous variables were presented as mean±standard deviations (SD) and categorical variables were presented as percentages. Independent t test was done to check any significant correlation of AIP with gender. Assumption of normality and equality of variances done by Shapiro Wilk test and Levene's test. The relation between AIP and HDL-C was done by F test (analysis of variances). The relation between triglyceride (TG) and AIP was checked by Kruskal-Wallis test. The correlation between AIP and cardiac troponin I was calculated by Spearman's rho correlation coefficient.

RESULTS

Data of total 50 patients were analyzed. Distribution of study participants according to gender shows that out of 50 myocardial infarction patients, 42 were male (84%) and 8 were female (Table 1).

Table 1: Distribution of study participants according to gender.

Gender	Frequency	Percentage (%)
Female	8	16
Male	42	84

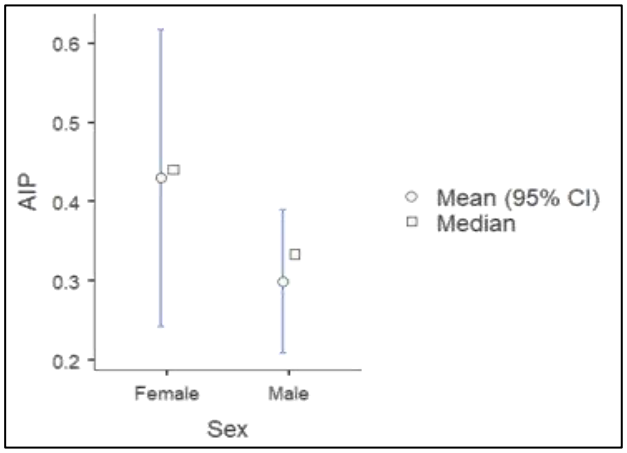

Figure 1: The test of assumption of normality and equality of variances done by Shapiro Wilk test and Levene's test.

Distribution of study participants according to religion shows 42% Hindu and 56% Muslim and others are 1%. (Table 2).

Table 3 shows distribution of study participants according to development of risk of atherosclerosis. We found that among the 50 myocardial infarction patients 33 fall in the high-risk group when their AIP was calculated (66 %), 5 falls in the intermediate risk group (10%) and 12 falls in the low-risk group (24%).

Table 2: Distribution of study participants according to religion.

Religions	Frequency	Percentage (%)
Hindu	21	42
Muslim	28	56
Others	1	2

Table 3: Distribution of study participants according to development of risk of atherosclerosis.

AIP risk groups	Frequency	Percentage (%)
High risk	33	66
Intermediate risk	5	10
Low risk	12	24

Table 4 shows descriptive statistics of different parameters. The participants are having mean random blood sugar of 138 (SD=94.5-187), mean serum creatinine is 1.13 (SD=0.890-1.61), mean triglyceride 139 (SD=25.3-40.1), mean HDL 31 (SD=10.9), mean troponin I 0.441 (SD=0.0968-3.23), and mean AIP 0.320 (SD=0.297).

Table 4: Descriptive statistics of different parameters.

Blood parameters	Mean	Standard deviation
Random blood sugar	138*	(94.5-187) *
Serum creatinine	1.13*	(0.890-1.61) *
Triglyceride	139*	(25.3-40.1) *
HDL	31	10.9
Trop I	0.441*	(0.0968-3.23) *
AIP	0.320	0.297

*Median and interquartile range (IQR)

The relation of AIP with gender is measured by independent t test where we got the p=0.114 (p<0.05 taken as significant) which is insignificant (Table 5).

Table 5: Gender and AIP.

Gender	Mean±SD	Test value (df)	P value
Female	0.430	1.14 (48)	0.114
Male	0.334		

*Independent t test, p<0.05 taken as a significant.

The relation between AIP and HDL is measured by F test (analysis of variances) and shows a p=0.005 (p<0.05 taken as significant) which is significant (Table 6).

The relation between triglyceride and AIP also looked for by Kruskal-Wallis test, where we found the p=0.00 (p<0.05 taken as significant) which is significant (Table 7).

Table 6: HDL and AIP.

Parameter	AIP	Mean±SD	df1, df2	P
HDL	High risk	27.5±9.14	4.98, 2.47**	0.005
	Intermediate risk	36.5±8.62		
	Low risk	38.2±12.3		

*ANOVA, ** F test value, p<0.05 taken as a significant.

Table 7: Triglyceride and AIP.

Parameter	AIP	Median (IQR)	df	P
Triglyceride	High risk	158 (129-186)	24.7 (2)	0
	Intermediate risk	107 (104-107)		
	Low risk	80.8 (59.3-97.7)		

*Kruskal-Wallis test, p<0.05 taken as a significant.

Lastly the correlation between AIP and troponin I is calculated by Spearman's rho correlation coefficient (Figure 2) which came 0.138 and a p=0.339 (p<0.05 taken as significant) which is insignificant (Table 8).

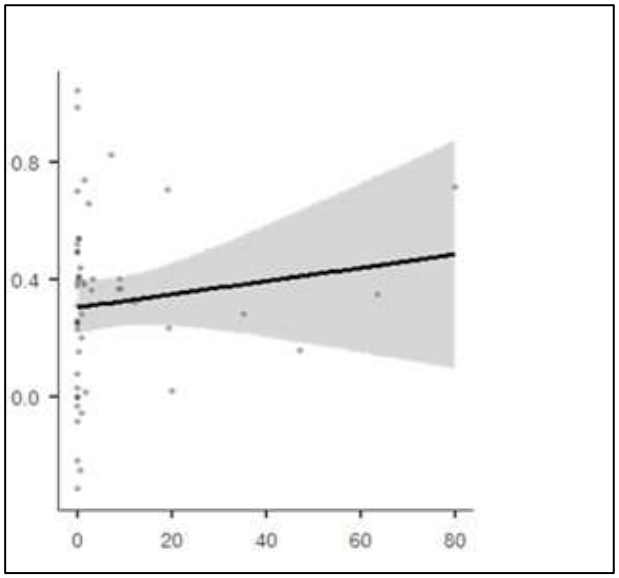

Figure 2: Spearman's rho correlation coefficient.

Table 8: Troponin I and AIP.

Variable	Spearman's rho correlation coefficient	P value
Trop I	0.138	0.339

*P<0.05 taken as a significant.

DISCUSSION

Many studies in past have shown the usefulness of AIP with respect to prediction of the risk of major cardiovascular diseases like myocardial infarction. Lafta in his study named "A comparative study for some atherogenic indices in sera of myocardial infarction, ischemic heart disease patients and control" shows that according to AIP the risk for myocardial infarction is more in developing cardiovascular disease than the established ischemic heart disease.[3] In that study he found that AIP is high in myocardial infarction patients than ischemic heart disease patients as compared to normal individuals. He mentioned in that study that calculating AIP from simple lipid profile can be a sensitive tool for risk stratification of cardio vascular diseases like myocardial infarction even when lipid profile is apparently normal. Cai et al showed in their study that AIP is a very strong independent predictor of CAD in Chinese Han population. Kazemi et al also established the relation of AIP with major cardiovascular events.[11,12]

In our study we found that 66 percent of the myocardial infarction patients have high AIP (>0.21) with mean AIP 0.320 and standard deviation of 0.297, with a male predominance of 84 percent with no significant dominancy based on specific religion. The mean AIP is 0.320 with a standard deviation of 0.297 which is also very significant. Rismawati et al obtained a mean AIP of 0.26 who did research on correlation of AIP with small dense LDL with degree of coronary artery stenosis in ACS patients.[6] They found that the most consistent associated parameter in coronary artery stenosis with ACS is small dense LDL which is also reflected by AIP. Guo et al in their study named "The sensibility of the new blood lipid indicator-atherogenic index of plasma (AIP) in menopausal women with coronary artery disease" found that AIP is an independent risk factor for coronary artery disease (CAD) even after adjustment of AIP for other risk factors for CAD.[12] In our study we got a mean HDL of 31 mg/dl with standard deviation 10.9 which is quite similar to the findings of Rismawati et al who got mean HDL 35.7 mg/dl with a standard deviation of 10.9.[6] We also found that low HDL is associated with high-risk group according to AIP (27.5±9.14) which gradually increases in the intermediate risk group (36.5±8.62) and highest in low-risk group (38.2±12.30). The AIP-HDL relationship is significant (p=0.005). It suggests low HDL bears the increase risk for myocardial infarction.

We had another observation in our study that serum triglyceride is more in myocardial infarction patients who are categorized as "high risk" according to AIP {158 (129-186)}, gradually in lower side in intermediate risk group {107 (104-107)} and lowest in low-risk group {80.8 (59.3-97.7)}. Our mean serum triglyceride was 139 mg/dl with a standard deviation of (25.3-40.1). This finding is quite similar to the observations of Bharwadj et al who did a study on association of AIP with coronary artery stenosis in ACS patients. Their observation was a mean triglyceride of 140.6 mg/dl with a standard deviation 6.3.[6] We got a significant positive correlation of AIP and triglyceride (p=0.00). These findings suggest that higher the serum triglyceride level, higher is the risk

for myocardial infarction as calculated by AIP. We didn't find any gender wise relationship with AIP (p=0.114).

In our study, findings are not suggestive of any relation of AIP with the severity of myocardial infarction with respect to value of troponin I (Spearman's rho correlation coefficient=0.138 and p=0.339).

Limitations

In our study other risk factors for myocardial infarction was not considered. Consideration of other risk factors will give more accurate result. Moreover, further studies with larger number of participants are required for confirmation of the findings.

CONCLUSION

According to the result of this study, a significant percentage (66%) of myocardial infarction patients had high AIP and were in the high-risk group (AIP>0.21). We also obtained significant association of high triglyceride and low HDL-C with AIP, both type of cases had high AIP in our study. We got a male predominance in myocardial infarction with no religion wise dominancy. Neither did we find any gender wise variation of AIP in myocardial infarction patients nor any relation of AIP with the severity of myocardial infarction with respect to the value of cardiac troponin I. As in India the treatment of cardiovascular diseases like myocardial infarction is very costly, by calculating AIP from fasting lipid profile, the high-risk patients can be easily identified and early interventions can be done to prevent the mortality and morbidly as well as it will increase the productivity of the country by cutting the huge treatment cost and by increasing the quality of life.

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