BMC Cardiovascular Disorders

RESEARCH

Open Access

The relationship between the atherogenic index of plasma and postoperative myocardial injury following non-cardiac surgery under general anaesthesia: a retrospective cohort study

Yuanjun Zhou[1], Liping Zhong[1], Yilin Liao[1] and Yuting Zhong[1*]

Abstract

Background The atherogenic index of plasma (AIP) is a reliable lipid marker associated with coronary artery stenosis (CAS) and cardiovascular events. However, the relationship between AIP and myocardial injury after non-cardiac surgery (MINS) remains insufficiently explored.

Methods This retrospective study included adult patients who underwent non-cardiac surgery under general anaesthesia. The primary exposure was preoperative AIP, with MINS as the primary outcome. The predictive accuracy of AIP for MINS was assessed using the area under the curve (AUC). Restricted cubic splines (RCS) were used to explore the potential nonlinear relationship between AIP and MINS. Logistic regression analysis was conducted to examine the association of AIP with MINS. Subgroup and interaction analyses were carried out across multiple factors, including age, gender, body mass index, medical history, and the type of surgery (emergency or elective).

Results The cohort consisted of 1,160 adult patients, with a median preoperative AIP of -0.05. The incidence of MINS was 7.9%. The AUC for AIP in predicting MINS was 0.719, surpassing the AUCs of triglycerides and high-density lipoprotein cholesterol (0.644 and 0.683, respectively). RCS analysis demonstrated a linear relationship between AIP and MINS (P for nonlinear = 0.165). Patients in the highest quartile of AIP had significantly higher odds of developing MINS than those in the lowest quartile (adjusted OR, 8.05; 95% confidence interval [CI], 3.44 to 18.80; $P < 0.001$). The results across most subgroups were consistent with the primary analysis, showing no significant interaction effects.

Conclusions A significant and independent linear relationship exists between preoperative AIP and the risk of MINS. As an economical and easily accessible lipid marker, AIP holds potential for preoperative screening of patients at risk of postoperative cardiovascular events.

Keywords Triglyceride, High-density lipoprotein cholesterol, Myocardial injury, Non-cardiac surgery, General anaesthesia

*Correspondence:
Yuting Zhong
mzhospitalytzhong@163.com
[1]Department of Anaesthesiology, Meizhou People's Hospital, 63 Huangtang Road, Meijiang District, Meizhou, Guangdong, China

Introduction

Postoperative mortality in patients aged over 45 years old is estimated at 1–2%, with myocardial ischaemic injury as a primary cause [1, 2]. Myocardial injury after non-cardiac surgery (MINS) is strongly associated with adverse cardiovascular events (ACEs) within 30 days postoperatively and a decreased long-term survival rate [3, 4]. Consequently, anaesthesiologists prioritise preoperative cardiac screening, including physical examinations, electrocardiograms (ECG), and echocardiograms. Patients presenting with chest pain or diagnosed coronary artery disease (CAD) may undergo preoperative coronary angiography to assess the status of coronary artery lesions. However, patients with preoperative coronary artery stenosis (CAS) who lack obvious clinical symptoms may go undetected, complicating the identification of those at high risk of MINS. Preoperative coronary angiography is not a mandatory examination; therefore, a simple and reliable marker to preoperatively identify patients with potential CAD would be of significant clinical value in reducing the incidence of MINS.

Dyslipidaemia is a major risk factor for the development of CAD. Triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) collectively influence the progression of atherosclerosis. However, a single lipid measure does not fully capture the complexity of coronary atherosclerosis. Dobiasova et al. introduced the concept of the atherogenic index of plasma (AIP), defined as the logarithm of the ratio of TG to HDL-C in 2001 [5]. AIP is significantly negatively associated with the particle size of LDL and has been closely associated with the severity of coronary atherosclerosis and stenosis [5], indirectly reflecting the condition of coronary artery lesions. Compared to individual lipid indicators, AIP has demonstrated greater accuracy in assessing coronary atherosclerosis [6–9]. It is regarded as one of the most reliable predictors of CAD and ACEs, offering superior predictive power over traditional atherogenic lipid profiles [7].

While the positive correlation between AIP and both CAS and subclinical CAD [10] is well recognised, research examining the relationship between preoperative AIP and MINS remains limited. Preoperative TG and HDL-C levels are readily available, leading us to hypothesise that an elevated preoperative AIP is positively associated with the incidence of MINS. Validating this hypothesis could offer anaesthesiologists a simple and effective means to identify patients with high-risk preoperatively, enabling timely interventions such as coronary angiography and pharmacological treatments.

This study aims to investigate the association between preoperative AIP and MINS, addressing a critical gap in current research and advancing perioperative cardiac protection strategies in clinical practice.

Materials and methods

Study design and ethics

This secondary analysis was based on the original data from a single-center retrospective cohort study initially conducted at Meizhou People's Hospital, a tertiary institution. The original study received ethical review and approval from the Institutional Ethics Committee (Approval Number: 2023-C-92) and was registered with the National Medical Registry (Registration Number: ChiCTR2400082834). Our secondary analysis has also been reviewed and approved by the same Institutional Ethics Committee (Approval Number: 2024-C-150). This secondary analysis adheres to the ethical principles outlined in the 1964 Declaration of Helsinki and its subsequent amendments. Given the retrospective nature of the study, the Ethics Committee waived the requirement for informed consent. This study did not involve any patient or public participation.

Patient cohort

The study population included inpatients aged ≥ 40 years who underwent non-cardiac surgery under general anaesthesia with endotracheal intubation at the Surgical and Anaesthesia Centre between January 2019 and December 2023. The exclusion criteria were as follows: (i) patients without available electronic medical records; (ii) patients who underwent low-risk surgeries, including outpatient, hysteroscopic, or superficial surgeries; (iii) patients who had multiple surgeries; (iv) patients without TG or HDL-C measurements within 1 week preoperatively; and (v) patients lacking essential baseline information, such as preoperative laboratory results and anaesthesia information.

Data collection

We extracted the predefined variables from the electronic medical, laboratory, and anaesthesia records systems. The variables included: (i) demographic characteristics, including age, gender, and body mass index (BMI), and smoking history; (ii) medication history within 1 week preoperatively, including rate-controlling drugs, aspirin, heparin, and statins; (iii) preoperative comorbidities, including congestive heart failure (CHF), history of stroke, peripheral vascular disease (PVD), atrial fibrillation (AF), hypertension, myocardial infarction (MI), chronic obstructive pulmonary disease (COPD), and diabetes; (iv) laboratory results within 1 week preoperatively, comprising TG, HDL-C, blood glucose (BG), haemoglobin (Hb), estimated glomerular filtration rate (eGFR), and LDL-C; and (v) anaesthesia and surgery information, including the American Society of Anaesthesiologists (ASA) physical status, surgical types, duration of surgery, duration of the heart rate to mean arterial pressure ratio (HMR) > 1, blood loss, and red blood cell

(RBC) infusion. Surgical types include neurosurgery, thoracic surgery, gastrointestinal surgery, foregut or hepatopancreatobiliary surgery, orthopedic or spine surgery, and other surgeries. Other Surgeries included gastrointestinal endoscopy, bronchoscopy, otolaryngology, gynecological, thyroid, peripheral vascular intervention, oral and maxillofacial, breast, plastic, and ENT (ear, nose, and throat) surgeries.

Exposure of interest

AIP was calculated using the formula: Log[TG (mg/dL) × HDL-C (mg/dL)/2] [5]. TG and HDL-C values used in the calculation were the last recorded preoperative measurements. We ensured that the units of TG and HDL-C were appropriately converted for the calculations.

Outcome

The primary outcome was defined as MINS. In clinical practice, postoperative cardiac troponin (cTn) monitoring is not yet a routine procedure and is typically conducted for patients at risk of MINS, rather than universally for all patients. Consequently, cTn testing was not standardised in this retrospective study. To ensure accurate identification of patients with MINS, all researchers collaboratively reviewed the medical records of all participants, including troponin levels, ECGs, and clinical notes. The diagnostic criteria for MINS were as follows [4]: (i) postoperative elevation of cTn within the first 30 days, with at least one cTn measurement exceeding the 99th percentile upper reference limit, presumed to be of ischemic etiology, or demonstrating a rise-and-fall pattern indicative of acute myocardial injury; (ii) myocardial injury attributed to an underlying ischaemic mechanism, such as an imbalance between oxygen supply and demand or atherothrombosis; (iii) exclusion of non-ischaemic causes of troponin elevation, such as pulmonary embolism or sepsis; and (iv) the potential absence of clinical symptoms of ischaemia due to sedation or analgesia, meaning that ischaemic features (including symptoms and ECG changes) are not required for diagnosis.

Statistical analysis

Patients were divided into quartiles based on preoperative AIP levels to explore potential dose-response relationships by comparing MINS risk across the spectrum of AIP levels. Using quartiles provides a clearer view of risk gradients and the relationship between AIP and MINS. Data that did not follow a normal distribution (as per the Kolmogorov-Smirnov test) were presented as medians with interquartile ranges (IQR) and compared using the Wilcoxon rank sum test for two-group comparisons and the Kruskal-Wallis rank sum test for multiple-group comparisons. Categorical data were presented as frequencies (n) and percentages (%) and were compared using Pearson's chi-squared test and Fisher's exact tests, as appropriate. The optimal AIP threshold for predicting MINS was determined using a receiver operating characteristic (ROC) curve. To further elucidate the linear relationship between continuous AIP and MINS, multivariable restricted cubic splines (RCS) were employed. This method enabled us to investigate the incremental change in the risk of MINS for each unit increase in AIP. Logistic regression analyses (both univariate and multivariate) were conducted, with results expressed as adjusted odds ratios (ORs) and 95% confidence intervals (CI). Potential confounders were selected based on variables with a *P*-value of <0.2 in univariable analysis between MINS and non-MINS groups (Supplementary Table S4), along with other clinically relevant variables. The models were adjusted for age, gender, BMI, Hb, eGFR, BG, CHF, AF, hypertension, the history of MI, diabetes, the history of stroke, PVD, history of heparin and statin use, duration of HMR >1, duration of anaesthesia, ASA classification, type of surgery, emergency surgery, and intraoperative RBC transfusion. Subgroup analysis was conducted to evaluate the association between the dichotomous AIP and MINS within specific population subgroups, aiming to identify individuals most likely to benefit from AIP surveillance. This binary approach facilitates the straightforward classification of patients into high-risk and low-risk groups based on their AIP values, making it more easily applicable to clinical decision-making processes. Subgroup analyses were conducted based on age (<65 or ≥ 65 years), gender, BMI (<24 or ≥ 24 kg/m^2), hypertension, history of MI, diabetes, and type of surgery.

Data analysis was performed using https://medsta.cn/, a free and publicly accessible data analysis platform developed based on R 4.2 (R Foundation, Austria). A *P*-value of <0.05 was considered statistically significant (two-tailed).

Results

Reporting guidelines

This study reported its findings in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [11].

Baseline characteristics

The study cohort comprised 1,160 patients who underwent general anaesthesia with endotracheal intubation (Fig. 1). Patients were categorised into four quartile groups based on preoperative AIP levels (Table 1). The overall median AIP was −0.05 (IQR, -0.23–0.14). The distribution of preoperative AIP values is illustrated in Supplementary Figure S1. The overall incidence of MINS was found to be 7.9% (92/1160), with the incidence increasing as AIP levels rose ($P<0.001$). The median age

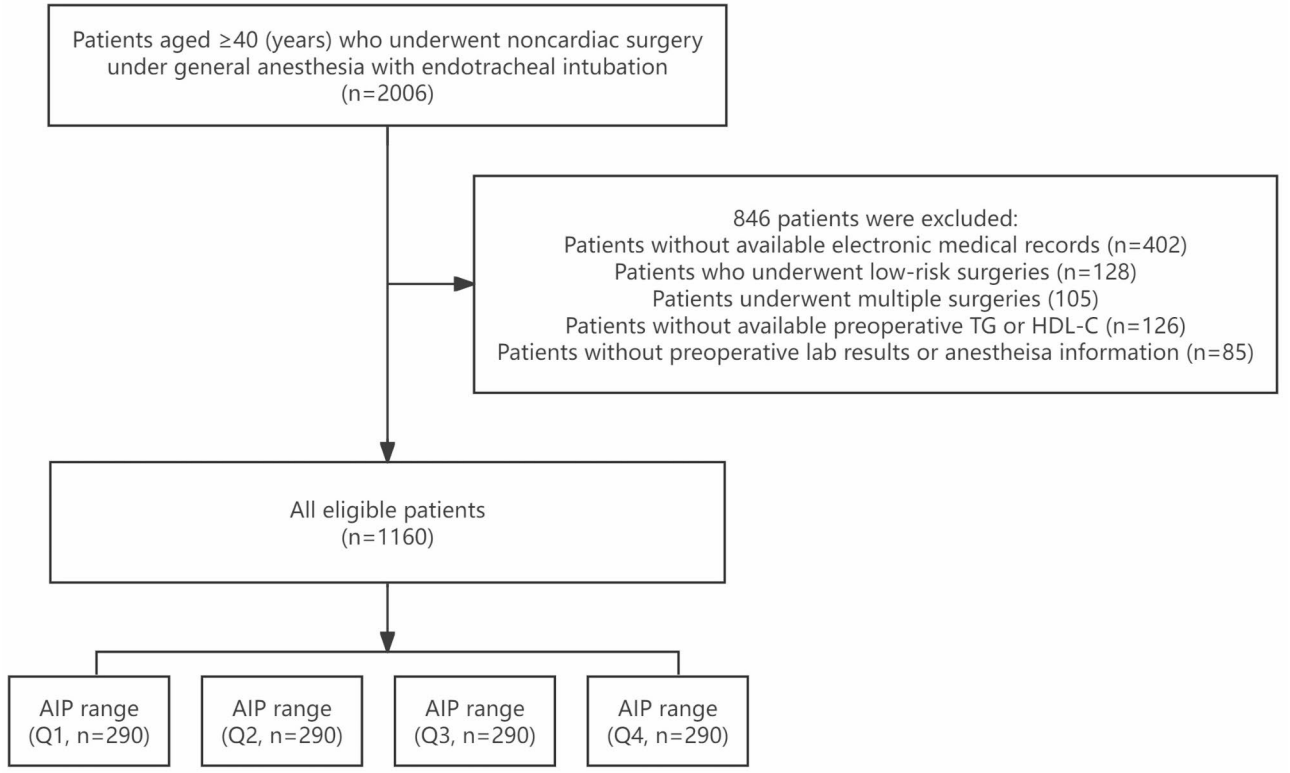

Fig. 1 Flowchart of patient inclusion and exclusion. Figure 1 Low-risk surgeries included outpatient procedures, hysteroscopic surgeries, and superficial surgeries. TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; AIP: atherogenic index of plasma. AIP quartile ranges: Q1 (-1.32 to -0.22), Q2 (-0.23 to -0.04), Q3 (-0.05 to 0.13), Q4 (0.14 to 0.80)

of the cohort was 64 years, with 41.6% of the patients being female. In the fourth quartile (Q4) group, patients exhibited a higher preoperative BMI, elevated BG levels, an increased prevalence of diabetes, and lower eGFR values. Among the various types of surgeries performed (Table 1 and Supplementary Figure S2), neurosurgery constituted the largest proportion at 21.8%, followed by thoracic surgery at 19.6%, and gastrointestinal surgery at 18.0%. Patients with higher AIP demonstrated a significantly increased MINS rate ($P<0.001$). Supplementary Table S3 details the magnitude of cTn elevation and the number of days postoperatively when troponin elevation was detected.

Univariate analysis

Detailed information regarding the MINS and non-MINS groups is presented in Supplementary Table S4. The median preoperative AIP in the MINS cohort was significantly higher than that in the non-MINS cohort (-0.07 vs. 0.12, respectively, $P<0.001$). The MINS cohort exhibited lower eGFR and Hb levels, while BG levels were notably higher (all $P<0.001$). Furthermore, this cohort demonstrated a greater prevalence of a history of MI, stroke, and PVD ($P<0.001$, $P=0.007$, $P=0.003$, respectively). The proportion of patients receiving preoperative heparin was significantly lower in the MINS cohort compared to the non-MINS cohort ($P<0.001$). Individuals in the MINS cohort also had a higher ASA classification and a longer duration of HMR > 1 ($P=0.045$, $P=0.001$, respectively).

Results of ROC

AIP demonstrated greater accuracy in predicting MINS compared to TG and HDL-C, with areas under the curve of 0.719, 0.644, and 0.683, respectively (Supplementary Figure S5). The optimal diagnostic threshold for AIP was determined to be -0.11, with a specificity of 90.2% and a sensitivity of 45.2%.

Results of multivariate logistic regression and RCS

In the adjusted model, each unit increase in the continuous AIP was positively correlated with an elevated risk of MINS (OR, 24.05; 95% CI, 9.17–63.06; $P<0.001$, Table 2). The difference in the risk of MINS between the Q2 and Q1 cohorts was not statistically significant ($P=0.135$, Table 2). However, both Q3 and Q4 cohorts exhibited a significant increase in the risk of MINS compared to the Q1 cohort ($P=0.002$, $P<0.001$, respectively, Table 2). A significant linear trend was evident in the adjusted model (P for trend < 0.001). The adjusted RCS analysis revealed

Table 1 Baseline characteristics of patients stratified by AIP index quartiles

	Overall, *N* = 1160	Q1, *N* = 290	Q2, *N* = 290	Q3, *N* = 290	Q4, *N* = 290	*P*
AIP index range		-1.32 to -0.22	-0.23 to -0.04	-0.05 to 0.13	0.14 to 0.80	/
AIP index quartile	-0.05 [-0.23, 0.14]	-0.36 [-0.48, -0.29]	-0.14 [-0.18, -0.10]	0.03 [-0.01, 0.08]	0.28 [0.21, 0.38]	< 0.001
TG (mmol/L)	1.16 [0.83, 1.62]	0.67 [0.55, 0.82]	1.01 [0.86, 1.15]	1.36 [1.15, 1.53]	2.04 [1.68, 2.54]	< 0.001
HDL-C	1.31 [1.09, 1.56]	1.61 [1.40, 1.89]	1.37 [1.22, 1.59]	1.25 [1.08, 1.41]	1.02 [0.87, 1.22]	< 0.001
Age (years)	64 [55, 72]	64 [54, 71]	65 [56, 72]	64 [55, 73]	62 [54, 72]	0.066
Sex, **woman**	483 (41.6)	128 (44.1)	119 (41.0)	120 (41.4)	116 (40.0)	0.773
BMI (kg/m^2)	22.2 [20.1, 24.6]	20.8 [19.1, 22.9]	22.3 [20.1, 24.5]	22.5 [20.2, 24.8]	23.3 [21.4, 25.2]	< 0.001
Smoking history	206 (17.8)	52 (18.0)	49 (16.90)	55 (19.0)	50 (17.2)	0.920
Preoperative medication history						
Rate-controlling drugs	63 (5.4)	9 (3.1)	21 (7.2)	18 (6.2)	15 (5.2)	0.152
Aspirin	37 (3.2)	10 (3.4)	9 (3.1)	10 (3.4)	8 (2.8)	0.959
Heparin	259 (22.3)	55 (19.0)	73 (25.2)	63 (21.7)	68 (23.4)	0.319
Statins	113 (9.7)	21 (7.2)	28 (9.7)	26 (9.0)	38 (13.1)	0.112
Preoperative medical history						
CHF	24 (2.1)	5 (1.7)	8 (2.8)	6 (2.1)	5 (1.7)	0.796
AF	11 (0.9)	5 (1.7)	2 (0.7)	4 (1.4)	0 (0.0)	0.116
Hypertension	238 (20.5)	45 (15.5)	62 (21.4)	61 (21.0)	70 (24.1)	0.073
History of MI	65 (5.6)	10 (3.4)	19 (6.6)	13 (4.5)	23 (7.9)	0.082
COPD	86 (7.4)	26 (9.0)	24 (8.3)	23 (7.9)	13 (4.5)	0.166
Diabetes	111 (9.6)	12 (4.1)	24 (8.3)	23 (7.9)	52 (17.9)	< 0.001
History of Stroke	66 (5.7)	14 (4.8)	17 (5.9)	14 (4.8)	21 (7.2)	0.548
PVD	77 (6.6)	14 (4.8)	21 (7.2)	20 (6.9)	22 (7.6)	0.541
Preoperative Lab results						
Haemoglobin (g/L)	128 [115, 138]	126 [114, 137]	127 [116, 137]	129 [116, 139]	128 [111, 139]	0.516
Glucose (mmol/L)	5.34 [4.71, 6.26]	5.23 [4.62, 5.99]	5.27 [4.67, 6.15]	5.26 [4.68, 6.19]	5.67 [4.94, 7.07]	< 0.001
eGFR (mL/min/1.73 m^2)	79 [69, 90]	83 [73, 91]	78 [69, 89]	77 [69, 89]	77 [66, 90]	< 0.001
Anaesthesia information						
ASA classification						0.833
I-II	115 (9.9)	30 (10.3)	32 (11.0)	27 (9.3)	26 (9.0)	
III	743 (64.1)	177 (61.0)	183 (63.1)	193 (66.6)	190 (65.5)	
IV-V	302 (26.0)	83 (28.6)	75 (25.9)	70 (24.1)	74 (25.5)	
Elective surgery	996 (85.9)	241 (83.1)	250 (86.2)	255 (87.9)	250 (86.2)	0.408
Duration of anaesthesia (mins)	200 [135, 265]	186 [131, 255]	195 [130, 260]	210 [140, 282]	205 [140, 265]	0.094
Duration of intraoperative HMR > 1 (mins)	40 [9, 90]	31 [6, 81]	47 [6, 99]	43 [11, 99]	40 [11, 93]	0.157
Surgical types						0.029
Neurosurgery	253 (21.8)	69 (23.8)	60 (20.7)	58 (20.0)	66 (22.8)	
Thoracic surgery	227 (19.6)	64 (22.1)	52 (17.9)	59 (20.3)	52 (17.9)	
Gastrointestinal surgery	209 (18.0)	44 (15.2)	42 (14.5)	56 (19.3)	67 (23.1)	
Foregut or hepatopancreatobiliary surgery	152 (13.1)	34 (11.7)	54 (18.6)	31 (10.7)	33 (11.4)	
Orthopedic or spine surgery	125 (10.8)	36 (12.4)	35 (12.1)	25 (8.6)	29 (10.0)	
Other surgeries	194 (16.7)	43 (14.8)	47 (16.2)	61 (21.0)	43 (14.8)	
Intraoperative bleeding (ml)	30 (20, 100)	30 (16, 100)	50 (20, 100)	42 (20, 100)	30 (20, 50)	0.155
Intraoperative RBC infusion	74 (6.4)	12 (4.1)	16 (5.5)	19 (6.6)	27 (9.3)	0.072
Postoperative cTn testing timing (days)	0.8 [0.7, 4.1]	0.8 [0.7,3.6]	0.8 [0.7, 4.0]	0.86 [0.7, 4.8]	0.84 [0.7, 4.0]	0.690

Continuous variables are presented as median [quartile], and categorical variables as n (proportion, %). Abbreviations AIP: atherogenic index of plasma; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; BMI: body mass index; rate-controlling drugs included β-blockers and calcium channel blockers; CHF: congestive heart failure; AF: atrial fibrillation; MI: myocardial infarction; COPD: chronic obstructive pulmonary disease; PVD: peripheral vascular disease; eGFR: estimated glomerular filtration rate (using the CKD-EPI formula); ASA: American Society of Anesthesiologists; HMR: heart rate to mean arterial pressure ratio; RBC: red blood cell; cTn: cardiac troponin. Other Surgeries: includes gastrointestinal endoscopy, bronchoscopy, otolaryngology, gynecological, thyroid, peripheral vascular intervention, oral and maxillofacial, breast, plastic, and ENT (ear, nose, and throat) surgeries

Table 2 Univariate and multivariate logistic regression analysis of AIP and MINS

	Model 1		Model 2		Model 3	
	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
Continuous AIP per unit	20.48(8.98–46.71)	< 0.001	15.10(6.20-36.78)	< 0.001	24.05 (9.17–63.07)	< 0.001
TyG index quartile group, (events/percentage)						
Q1, 8 (2.8)	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2, 14 (4.8)	1.79 (0.77–4.33)	0.198	1.68 (0.68–4.19)	0.263	2.03 (0.80–5.12)	0.135
Q3, 26 (9.0)	3.47(1.54–7.80)	0.003	3.32(1.43–7.69)	0.005	3.91(1.65–9.24)	0.002
Q4, 44 (15.2)	6.31(2.91–13.65)	< 0.001	5.39(2.40–12.10)	< 0.001	8.05(3.44–18.80)	< 0.001
P < 0.001	P_{trend} <0.001		P_{trend} <0.001		P_{trend} <0.001	

AIP quartile range: Q1 (-1.32 to -0.22), Q2 (-0.23 to -0.04), Q3 (-0.05 to 0.13), Q4 (0.14 to 0.80). Model 1: Crude. Model 2: Adjust: history of MI, PVD, stroke, history of heparin use, eGFR, Hb, BG, ASA classification, emergency surgery, duration of HMR > 1, intraoperative RBC transfusion. Model 3: Adjust: age, gender, BMI, Hb, eGFR, BG, CHF, AF, hypertension, history of MI, diabetes, history of stroke, PVD, history of heparin and statin use, duration of HMR > 1, duration of anaesthesia, ASA classification, type of surgery, emergency surgery, and intraoperative RBC transfusion. Abbreviations OR: Odds Ratio; CI: Confidence Interval; AIP: atherogenic index of plasma; MINS: myocardial injury after non-cardiac surgery; OR: odds ratio; CI: confidence interval. BMI: body mass index; Hb: heamoglobin; eGFR: estimated glomerular filtration rate; BG: blood glucose; CHF: congestive heart failure; AF: atrial fibrillation; MI: myocardial infarction; PVD: peripheral vascular disease; HMR: heart rate to mean blood pressure ratio; ASA: American Society of Anesthesiologists; RBC: red blood cell

a direct, linear increase in the risk of MINS alongside rising preoperative AIP levels (P for nonlinear = 0.165; Fig. 2). Accordingly, we employed multi-model linear regression analysis to investigate the linear relationship between preoperative AIP and postoperative cTn. The results are presented in Supplementary Table S6.

Subgroup analysis

To enhance clinical applicability, patients were categorised into two groups based on the optimal cutoff value of AIP (≤-0.11 vs. >-0.11) in order to examine heterogeneity across subgroups (Fig. 3 and Supplementary Table S7). The majority of results from the subgroup analyses were consistent with those of the primary analysis, and no significant interactions were identified between the subgroups. An elevated AIP was significantly associated with MINS in the non-diabetic subgroup (OR, 6.84; 95% CI, 3.28–4.28; $P < 0.001$) and the non-MI subgroup (OR, 7.49; 95% CI, 3.34–16.82; $P < 0.001$). However, this association was not observed in the subgroup with a BMI > 24 kg/m^2 (OR, 2.92; 95% CI, 0.73–11.74, $P = 0.131$) or in the thoracic surgery subgroup (OR, 4.42; 95% CI, 0.75–26.08; $P = 0.101$).

Discussion

The objective of this retrospective study was to investigate the potential application of preoperative AIP as a predictor for MINS. After adjusting for various risk factors, the results demonstrated a significant, independent linear relationship between elevated preoperative AIP and the risk of MINS. These findings provide a novel perspective on the assessment of the preoperative risk of MINS. To the best of our knowledge, previous studies have rarely explored the association between preoperative AIP and postoperative cardiovascular events.

Numerous studies have established a significant correlation between AIP and ACEs, metabolic disorders, and chronic kidney disease [12–16]. However, research specifically focusing on the association between preoperative AIP and MINS remains limited. Earlier investigations have indicated that poor metabolic conditions, such as insulin resistance, are closely associated with an increased risk of CAD, ACEs, and cardiac dysfunction [17–19]. Our findings further elucidate a direct linear relationship between preoperative AIP and MINS, underscoring its efficacy as a risk assessment tool for patients undergoing surgery. AIP has been identified as a predictive factor for CAD, surpassing traditional markers such as LDL-C, HDL-C, total cholesterol, and TG [7]. Moreover, due to its comprehensive reflection of lipid distribution and potential risk for atherosclerosis, AIP demonstrated superior predictive power for MINS compared to TG or HDL-C alone in our study. In subgroups without obesity or a history of MI, diabetes, or hypertension, the association between AIP and MINS remained significant, confirming its broad applicability. However, in patients with obesity and those undergoing thoracic surgery, the predictive capacity of AIP was diminished, likely due to the inherent risk for cardiovascular events associated with obesity and the complex haemodynamic fluctuations during thoracic procedures. Various cytokines released by adipose tissue play a significant role in providing anti-inflammatory protection and maintaining endothelial homeostasis [20]. Although the overall incidence of MINS is consistent with previous studies, the incidence in the group with AIP < -0.11 is lower (Fig. 3), likely due to the predefined patient inclusion criteria and its low prevalence. This low event rate highlights the need for caution when interpreting the results of the subgroup analysis. Consequently, for patients with high risk, a personalised prediction strategy that incorporates multiple risk assessment factors is essential for enhancing the accuracy of risk prediction for MINS. Another notable strength of this study is the inclusion of HMR as a haemodynamic parameter. HMR is highly significant because intraoperative hypotension frequently coincides

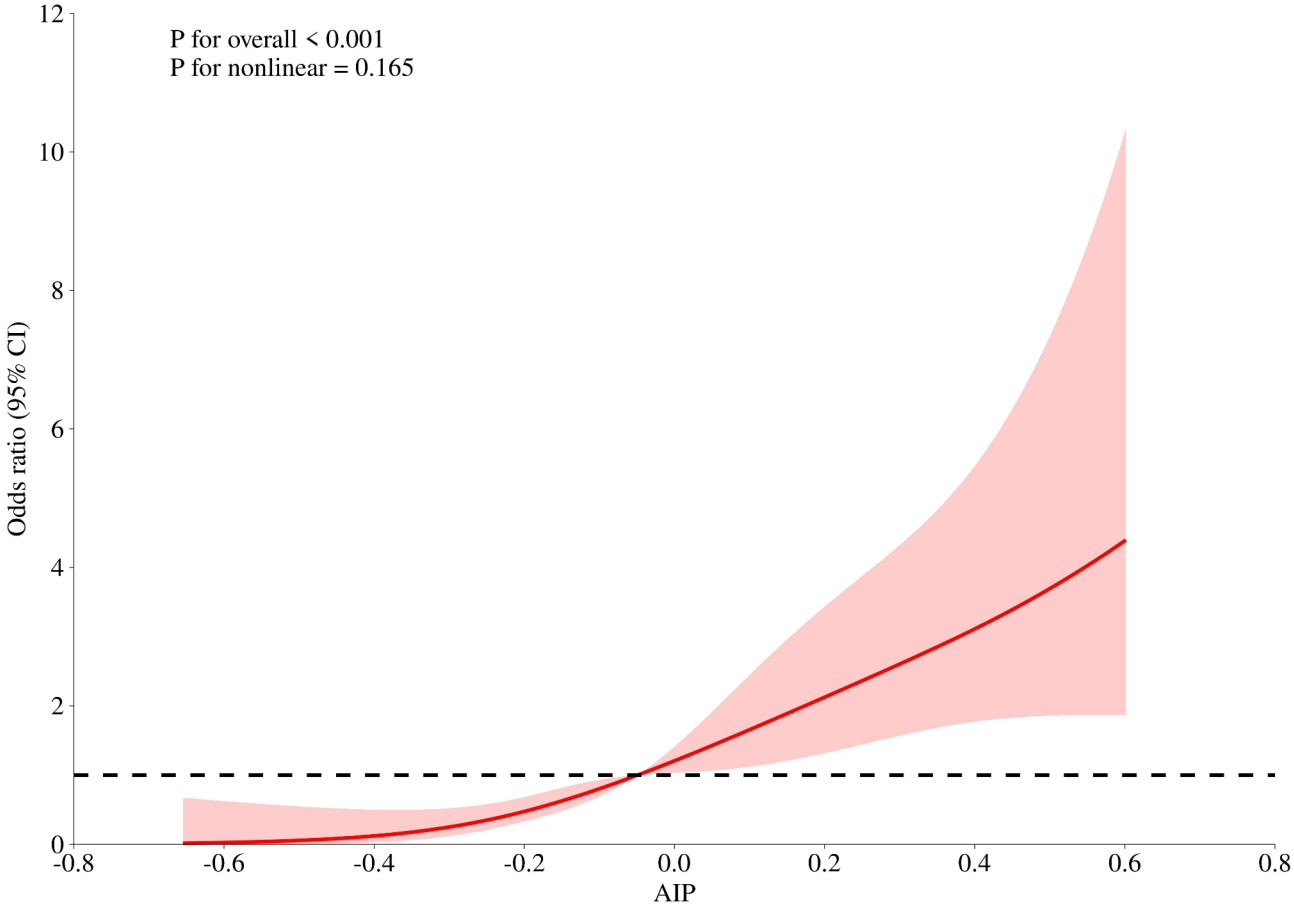

Fig. 2 Adjusted restricted cubic splines illustrating the relationship between preoperative AIP and MINS. Figure 2 AIP: atherogenic index of plasma. The solid line represents the association between preoperative AIP and MINS, while the horizontal dashed line denotes an odds ratio of 1. The shaded area indicates the 95% confidence interval for the odds ratio. The RCS model was adjusted for age, sex, BMI, haemoglobin (Hb), estimated glomerular filtration rate (eGFR), blood glucose (BG), congestive heart failure (CHF), atrial fibrillation (AF), hypertension, history of myocardial infarction (MI), diabetes, history of stroke, peripheral vascular disease (PVD), history of heparin and statin use, duration of HMR > 1, duration of anaesthesia, ASA classification, type of surgery, emergency surgery, and intraoperative RBC transfusion

with tachycardia, and it offers a more effective means of guiding the management of MBP, HR, fluid volume, and vasopressor use. To further reinforce the robustness of the primary findings, we have included the results of incorporating MBP as a haemodynamic parameter in the model analysis in the Supplementary Table S8.

AIP is calculated based on TG and HDL-C levels. It serves as a surrogate marker for small dense LDL (sdLDL) particles, which are negatively correlated with the particle size of LDL-C [8]. Elevated levels of TG, sdLDL, and reduced HDL-C collectively contribute to lipid deposition in the vascular wall, thereby inducing atherosclerosis and the formation of thrombosis [21]. TG plays a crucial role in the transport of lipoproteins between the arterial wall and endothelial cells and is associated with a greater number of LDL particles, making it more significant in the development of atherosclerosis than LDL-C alone [22, 23]. Increased levels of TG elevate the content of oxidised LDL-C in plasma, decrease the levels of HDL-C, and induce oxidative stress, mitochondrial dysfunction, and inflammatory responses—all critical factors in the development of coronary atherosclerosis [24, 25]. HDL-C possesses antioxidant properties, inhibits vascular inflammation, and enhances endothelial function [26, 27]. An elevated AIP indicates increased sdLDL and inflammatory responses, which promote the progression of atherosclerotic plaques [10, 28, 29]. SdLDL is more likely to infiltrate the arterial wall and tends to oxidise, facilitating its deposition in the extracellular matrix of the vascular wall and initiating the adhesion and aggregation of cholesterol, ultimately leading to atherosclerosis [30–32]. Furthermore, sdLDL can easily penetrate endothelial cells, causing oxidative damage and significantly enhancing its role in promoting coronary atherosclerosis [33–35]. SdLDL demonstrates higher predictive accuracy for ACEs compared to TG and HDL-C [9, 36], although its high detection cost limits its clinical application. As an indirect indicator of particle diameter of sdLDL, AIP

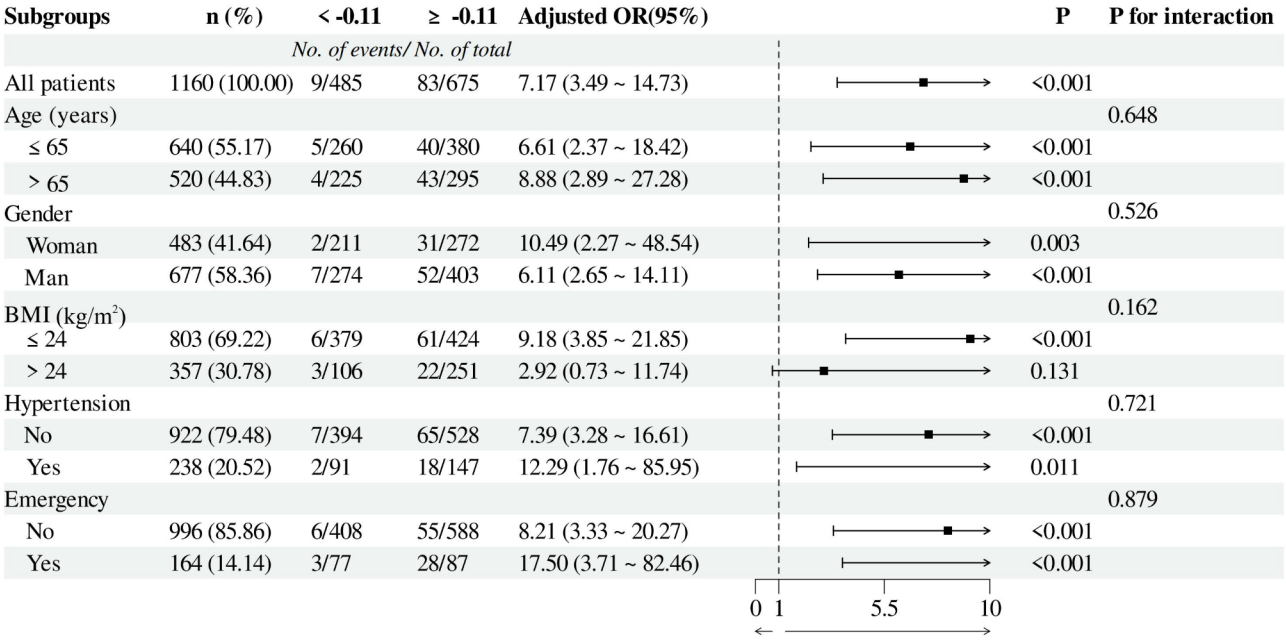

Fig. 3 Subgroup analysis. Figure 3 Cohorts were divided into two groups based on the optimal cutoff value of AIP (< -0.11 vs. ≥ -0.11). OR: odds ratio; CI: confidence interval; BMI: body mass index. The odds ratios were adjusted for age, sex, BMI, haemoglobin (Hb), estimated glomerular filtration rate (eGFR), blood glucose (BG), congestive heart failure (CHF), atrial fibrillation (AF), hypertension, history of myocardial infarction (MI), diabetes, history of stroke, peripheral vascular disease (PVD), history of heparin and statin use, duration of HMR > 1, duration of anaesthesia, ASA classification, type of surgery, emergency surgery, and intraoperative RBC transfusion

is significant for assessing the severity of coronary artery lesions and subclinical CAD [10, 37]. Additionally, AIP is closely associated with blood pressure, inflammatory responses, oxidative stress, and other risk factors for coronary atherosclerosis [38, 39].

An elevated AIP can predict chronic total occlusion of coronary angiography [40]. Even among healthy adults, AIP is associated with CAS [41]. Increased AIP may indirectly elevate the risk of MINS by promoting the development of CAD and its associated inflammatory and oxidative stress responses. As CAS worsens and myocardial blood perfusion diminishes, the compromised coronary arteries may struggle to meet the oxygen demands of the myocardium during surgical procedures, potentially leading to myocardial ischaemia and hypoxia. Furthermore, surgical-related inflammatory responses and oxidative stress may exacerbate myocardial damage. The interplay of inflammatory responses, oxidative stress, and endothelial dysfunction plays a synergistic role in the elevation of AIP, CAD, and postoperative MI, forming the core mechanism of this complex pathological process. These findings underscore the importance of preoperative lipid management in preventing postoperative ACEs. While preoperative coronary angiography is not a mandatory examination for risk assessment, preoperative AIP has the potential to evaluate the condition of the coronary arteries. For patients with elevated preoperative AIP, it may be prudent to assess the coronary artery status and consider the use of lipid-lowering medications. However, since preoperative lipid testing may not be routinely performed in certain medical regions, it is important to consider the general applicability of this study's findings. The results suggest that preoperative lipid testing could play a valuable role in assessing the risk of MINS and identifying patients who might benefit from preoperative cardiovascular optimisation. This highlights the importance of incorporating preoperative lipid testing in comparable medical settings to enhance the assessment and management of perioperative ACEs.

The relationship between AIP, lipid-lowering drugs, and MINS remains an area of interest. In this study, only a portion of patients with lipid disorders may have received lipid-lowering treatment before surgery. The lack of high-quality data on statins means their efficacy in preventing and treating MINS remains controversial [4]. Preoperative statin use has been associated with a reduced risk of MINS [42]. However, another study found that administering a loading dose of atorvastatin to high-risk patients before surgery did not reduce the incidence of MINS [43]. Furthermore, patients with MINS who did not receive postoperative statins treatment faced an increased risk of ACEs within one year compared to patients without MINS [44]. For those with perioperative MI, statin use at discharge has been linked to lower 30-day mortality [45]. Patients with perioperative MI exhibit a higher incidence of atherosclerotic

vascular disease [46–48], which is strongly correlated with elevated AIP and highlights the potential necessity of preoperative lipid testing. Given the potential benefits of statins, it seems prudent to consider statin therapy for patients with atherosclerotic disease and high AIP to mitigate ACEs. However, prospective studies are needed to further evaluate the interplay between statin use, AIP, and postoperative ACEs.

This study has several limitations. Firstly, due to the low rate of MINS in patients aged < 40 years, only patients aged ≥ 40 years were included. This approach may limit the generalisability of the findings across the entire age range. Secondly, the retrospective nature of the study imposes limitations on the standardisation of data collection and the controlling on the timing of postoperative interventions, particularly regarding the timing of preoperative lipid testing, postoperative cTn testing, and postoperative lipid management. As postoperative cTn testing was not universally conducted, but rather selectively performed on high-risk patients or when clinical concerns arose, this may have affected the general applicability of our study findings. Thirdly, the overall incidence of MINS aligns with findings from previous studies, however, the low prevalence of MINS underscores the need for caution when interpreting the results of the subgroup analysis. Given the limited number of positive events, the event-to-variable ratio fell below the traditional threshold. Although we validated the stability of our findings using multiple multivariable models (Supplementary Table S9), further validation in larger cohorts is necessary to address potential limitations related to the event-to-variable ratio. Lastly, the existing medical records indicated that only a subset of patients underwent troponin testing and were identified. While this reflects real-world clinical practice, it may introduce bias. In the future, prospective studies should adopt a more standardised cTn testing protocol to enable real-time monitoring.

Conclusion

This retrospective study identified a significant independent linear relationship between preoperative AIP and MINS. As a readily accessible marker, AIP can effectively stratify the risk of postoperative ACEs. However, well-designed prospective studies are necessary to elucidate its role in preoperative intervention strategies and to establish the optimal management protocols.

Abbreviations

MINS	Myocardial Injury after Non-Cardiac Surgery
ACEs	Adverse Cardiovascular Events
ECG	Electrocardiogram
CAD	Coronary Artery Disease
CAS	Coronary Artery Stenosis
TG	Triglycerides
LDL-C	Low-Density Lipoprotein Cholesterol
HDL-C	High-Density Lipoprotein Cholesterol
AIP	Atherogenic Index of Plasma
BMI	Body Mass Index
MI	Myocardial infarction
CHF	Congestive Heart Failure
PVD	Peripheral Vascular Disease
AF	Atrial Fibrillation
COPD	Chronic Obstructive Pulmonary Disease
BG	Blood Glucose
Hb	Haemoglobin
eGFR	Estimated Glomerular Filtration Rate
ASA	American Society of Anaesthesiologists
HMR	Heart Rate to Mean Arterial Pressure Ratio
RBC	Red Blood Cell
ENT	Ear, Nose, and Throat
cTn	Cardiac troponin
ROC	Receiver Operating Characteristic
RCS	Restricted Cubic Splines
OR	Odds Ratio
CI	Confidence Interval
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
sdLDL	Small Dense Low-density Lipoprotein

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04534-w.

Supplementary Material 1

Acknowledgements

We would like to thank EditChecks (https://editchecks.com.cn/) for providing linguistic assistance during the preparation of this manuscript. We would also like to thank Mr. Weiming Chen (chenweiming2@mzrmyy.com) and Mrs. Fei Liang (liangfei@mzrmyy.com) for their assistance in data processing.

Author contributions

Yuanjun Zhou: study design, data collection and examination, data analysis, and manuscript drafting, manuscript revision; Liping Zhong: data examination and data analysis; Yilin Liao: data examination and analysis and the supervision of the study process, manuscript revision; Yuting Zhong: study design, data collection, and examination, data analysis, manuscript drafting, manuscript revision and supervision of the study process. All authors revised the manuscript and approved the submission.

Funding

The research did not receive any external funding.

Data availability

The corresponding author can grant data access to this study upon request.

Declarations

Ethics approval and consent to participate

This secondary analysis was based on the original data from a single-center retrospective cohort study initially conducted at Meizhou People's Hospital, a tertiary institution. The original study received ethical review and approval from the Institutional Ethics Committee (Approval Number: 2023-C-92) and was registered with the National Medical Registry (Registration Number: ChiCTR2400082834). Our secondary analysis has also been reviewed and approved by the same Institutional Ethics Committee (Approval Number: 2024-C-150). This secondary analysis adheres to the ethical principles outlined in the 1964 Declaration of Helsinki and its subsequent amendments. Given the retrospective nature of the study, the Ethics Committee waived the requirement for informed consent.

Consent for publication

Not applicable.

Patient and public involvement
This study did not involve any members of the public or patients.

Competing interests
The authors declare no competing interests.

Received: 18 September 2024 / Accepted: 28 January 2025
Published online: 03 February 2025

References

1. Bartels K, Karhausen J, Clambey ET, Grenz A, Eltzschig HK. Perioperative organ injury. Anesthesiology. 2013;119(6):1474–89.
2. Writing Committee for the VSI, Devereaux PJ, Biccard BM, Sigamani A, Xavier D, Chan MTV, et al. Association of Postoperative High-Sensitivity troponin levels with myocardial Injury and 30-Day mortality among patients undergoing noncardiac surgery. JAMA. 2017;317(16):1642–51.
3. Devereaux PJ, Szczeklik W. Myocardial injury after non-cardiac surgery: diagnosis and management. Eur Heart J. 2020;41(32):3083–91.
4. Ruetzler K, Smilowitz NR, Berger JS, Devereaux PJ, Maron BA, Newby LK, et al. Diagnosis and management of patients with myocardial Injury after noncardiac surgery: a Scientific Statement from the American Heart Association. Circulation. 2021;144(19):e287–305.
5. Dobiasova M, Frohlich J. The plasma parameter log (TG/HDL-C) as an atherogenic index: correlation with lipoprotein particle size and esterification rate in apob-lipoprotein-depleted plasma (FER(HDL)). Clin Biochem. 2001;34(7):583–8.
6. Cai G, Liu W, Lv S, Wang X, Guo Y, Yan Z, et al. Gender-specific associations between atherogenic index of plasma and the presence and severity of acute coronary syndrome in very young adults: a hospital-based observational study. Lipids Health Dis. 2019;18(1):99.
7. Wu TT, Gao Y, Zheng YY, Ma YT, Xie X. Atherogenic index of plasma (AIP): a novel predictive indicator for the coronary artery disease in postmenopausal women. Lipids Health Dis. 2018;17(1):197.
8. Guo Q, Zhou S, Feng X, Yang J, Qiao J, Zhao Y, et al. The sensibility of the new blood lipid indicator–atherogenic index of plasma (AIP) in menopausal women with coronary artery disease. Lipids Health Dis. 2020;19(1):27.
9. Bendzala M, Sabaka P, Caprnda M, Komornikova A, Bisahova M, Baneszova R, et al. Atherogenic index of plasma is positively associated with the risk of all-cause death in elderly women: a 10-year follow-up. Wien Klin Wochenschr. 2017;129(21–22):793–8.
10. Si Y, Fan W, Han C, Liu J, Sun L. Atherogenic index of plasma, triglyceride-glucose index and monocyte-to-lymphocyte ratio for Predicting Subclinical Coronary Artery Disease. Am J Med Sci. 2021;362(3):285–90.
11. von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenbroucke JP, et al. The strengthening the reporting of Observational studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. J Clin Epidemiol. 2008;61(4):344–9.
12. Song P, Xu L, Xu J, Zhang HQ, Yu CX, Guan QB, et al. Atherogenic Index of Plasma is Associated with Body Fat Level in type 2 diabetes Mellitus patients. Curr Vasc Pharmacol. 2018;16(6):589–95.
13. Li YW, Kao TW, Chang PK, Chen WL, Wu LW. Atherogenic index of plasma as predictors for metabolic syndrome, hypertension and diabetes mellitus in Taiwan citizens: a 9-year longitudinal study. Sci Rep. 2021;11(1):9900.
14. Zhou Y, Shang X. Usefulness of atherogenic index of plasma for estimating reduced eGFR risk: insights from the national health and nutrition examination survey. Postgrad Med. 2021;133(3):278–85.
15. Yuan Y, Hu JW, Wang Y, Wang KK, Zheng WL, Chu C, et al. Association between atherogenic index of plasma and subclinical renal damage over a 12-year follow-up: Hanzhong adolescent hypertension study. Eur J Clin Nutr. 2020;74(2):278–84.
16. Shen SW, Lu Y, Li F, Yang CJ, Feng YB, Li HW, et al. Atherogenic index of plasma is an effective index for estimating abdominal obesity. Lipids Health Dis. 2018;17(1):11.
17. da Silva A, Caldas APS, Hermsdorff HHM, Bersch-Ferreira AC, Torreglosa CR, Weber B, et al. Triglyceride-glucose index is associated with symptomatic coronary artery disease in patients in secondary care. Cardiovasc Diabetol. 2019;18(1):89.
18. Jin JL, Cao YX, Wu LG, You XD, Guo YL, Wu NQ, et al. Triglyceride glucose index for predicting cardiovascular outcomes in patients with coronary artery disease. J Thorac Dis. 2018;10(11):6137–46.
19. Novo G, Manno G, Russo R, Buccheri D, Dell'Oglio S, Morreale P, et al. Impact of insulin resistance on cardiac and vascular function. Int J Cardiol. 2016;221:1095–9.
20. Valentijn TM, Galal W, Tjeertes EK, Hoeks SE, Verhagen HJ, Stolker RJ. The obesity paradox in the surgical population. Surgeon. 2013;11(3):169–76.
21. Dobiásová M, Frohlich J. [The new atherogenic plasma index reflects the triglyceride and HDL-cholesterol ratio, the lipoprotein particle size and the cholesterol esterification rate: changes during lipanor therapy]. Vnitr Lek. 2000;46(3):152–6.
22. Nordestgaard BG, Varbo A. Triglycerides and cardiovascular disease. Lancet. 2014;384(9943):626–35.
23. Peng J, Luo F, Ruan G, Peng R, Li X. Hypertriglyceridemia and atherosclerosis. Lipids Health Dis. 2017;16(1):233.
24. Chang W, Zhu F, Zheng H, Zhou Z, Miao P, Zhao L, et al. Glucagon-like peptide-1 receptor agonist dulaglutide prevents ox-LDL-induced adhesion of monocytes to human endothelial cells: an implication in the treatment of atherosclerosis. Mol Immunol. 2019;116:73–9.
25. Reiche ME, den Toom M, Willemsen L, van Os B, Gijbels MJJ, Gerdes N, et al. Deficiency of T cell CD40L has minor beneficial effects on obesity-induced metabolic dysfunction. BMJ Open Diabetes Res Care. 2019;7(1):e000829.
26. Wong NKP, Nicholls SJ, Tan JTM, Bursill CA. The role of high-density lipoproteins in diabetes and its vascular complications. Int J Mol Sci. 2018;19(6).
27. Poti F, Simoni M, Nofer JR. Atheroprotective role of high-density lipoprotein (HDL)-associated sphingosine-1-phosphate (S1P). Cardiovasc Res. 2014;103(3):395–404.
28. Farmer JA, Gotto AM. Jr. Dyslipidemia and the vulnerable plaque. Prog Cardiovasc Dis. 2002;44(6):415–28.
29. Cure E, Cumhur Cure M. Comment on the relationship between atherogenic index of plasma and no–reflow in patients with acute ST–segment elevation myocardial infarction who underwent primary percutaneous coronary intervention. Int J Cardiovasc Imaging. 2020;36(5):797–8.
30. Sharma P, Purohit P, Gupta R. Cardiac risk factors in descendants of parents with history of coronary artery disease (CAD): an evaluation focusing on small dense low density lipoprotein cholesterol (sdLDLc) and high density lipoprotein cholesterol (HDLc). Indian J Biochem Biophys. 2013;50(5):453–61.
31. Nishikura T, Koba S, Yokota Y, Hirano T, Tsunoda F, Shoji M, et al. Elevated small dense low-density lipoprotein cholesterol as a predictor for future cardiovascular events in patients with stable coronary artery disease. J Atheroscler Thromb. 2014;21(8):755–67.
32. Lamarche B, Tchernof A, Moorjani S, Cantin B, Dagenais GR, Lupien PJ, et al. Small, dense low-density lipoprotein particles as a predictor of the risk of ischemic heart disease in men. Prospective results from the Quebec Cardiovascular Study. Circulation. 1997;95(1):69–75.
33. Rizos CV, Elisaf MS, Liberopoulos EN. Effects of thyroid dysfunction on lipid profile. Open Cardiovasc Med J. 2011;5:76–84.
34. Ivanova EA, Myasoedova VA, Melnichenko AA, Grechko AV, Orekhov AN. Small dense low-density lipoprotein as Biomarker for atherosclerotic diseases. Oxid Med Cell Longev. 2017;2017:1273042.
35. Dobiasova M, Frohlich J, Sedova M, Cheung MC, Brown BG. Cholesterol esterification and atherogenic index of plasma correlate with lipoprotein size and findings on coronary angiography. J Lipid Res. 2011;52(3):566–71.
36. Cai G, Shi G, Xue S, Lu W. The atherogenic index of plasma is a strong and independent predictor for coronary artery disease in the Chinese Han population. Med (Baltim). 2017;96(37):e8058.
37. Dobiasova M. Atherogenic index of plasma [log(triglycerides/HDL-cholesterol)]: theoretical and practical implications. Clin Chem. 2004;50(7):1113–5.
38. Fernandez-Macias JC, Ochoa-Martinez AC, Varela-Silva JA, Perez-Maldonado IN. Atherogenic index of plasma: Novel Predictive Biomarker for Cardiovascular illnesses. Arch Med Res. 2019;50(5):285–94.
39. Hussain A, Ballantyne CM, Saeed A, Virani SS. Triglycerides and ASCVD risk reduction: recent insights and future directions. Curr Atheroscler Rep. 2020;22(7):25.
40. Liu T, Liu J, Wu Z, Lv Y, Li W. Predictive value of the atherogenic index of plasma for chronic total occlusion before coronary angiography. Clin Cardiol. 2021;44(4):518–25.
41. Nam JS, Kim MK, Park K, Choi A, Kang S, Ahn CW, et al. The plasma atherogenic index is an independent predictor of arterial stiffness in healthy koreans. Angiology. 2022;73(6):514–9.
42. Berwanger O, Le Manach Y, Suzumura EA, Biccard B, Srinathan SK, Szczeklik W, et al. Association between pre-operative statin use and major cardiovascular complications among patients undergoing non-cardiac surgery: the VISION study. Eur Heart J. 2016;37(2):177–85.

43. Berwanger O, de Barros ESPG, Barbosa RR, Precoma DB, Figueiredo EL, Hajjar LA, et al. Atorvastatin for high-risk statin-naive patients undergoing noncardiac surgery: the lowering the risk of operative complications using atorvastatin loading dose (LOAD) randomized trial. Am Heart J. 2017;184:88–96.
44. Foucrier A, Rodseth R, Aissaoui M, Ibanes C, Goarin JP, Landais P, et al. The long-term impact of early cardiovascular therapy intensification for postoperative troponin elevation after major vascular surgery. Anesth Analg. 2014;119(5):1053–63.
45. Devereaux PJ, Xavier D, Pogue J, Guyatt G, Sigamani A, Garutti I, et al. Characteristics and short-term prognosis of perioperative myocardial infarction in patients undergoing noncardiac surgery: a cohort study. Ann Intern Med. 2011;154(8):523–8.
46. Parashar A, Agarwal S, Krishnaswamy A, Sud K, Poddar KL, Bassi M, et al. Percutaneous intervention for myocardial infarction after noncardiac surgery: patient characteristics and outcomes. J Am Coll Cardiol. 2016;68(4):329–38.
47. Gualandro DM, Campos CA, Calderaro D, Yu PC, Marques AC, Pastana AF, et al. Coronary plaque rupture in patients with myocardial infarction after noncardiac surgery: frequent and dangerous. Atherosclerosis. 2012;222(1):191–5.
48. Duvall WL, Sealove B, Pungoti C, Katz D, Moreno P, Kim M. Angiographic investigation of the pathophysiology of perioperative myocardial infarction. Catheter Cardiovasc Interv. 2012;80(5):768–76.

Publisher's note