Journal of Cardiothoracic Surgery

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The role of long non-coding RNA A2M-AS1 in early diagnosis and prognosis evaluation of acute myocardial infarction

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Abstract

Aim The objective was to assess the clinical efficacy of long non-coding RNA (lncRNA) alpha-2-macroglobulin-antisense 1 (A2M-AS1) in acute myocardial infarction (AMI).

Methods One hundred patients with AMI and eighty patients with chest pain were recruited in the case-control study. A2M-AS1 expression was examined by quantitative real-time polymerase chain reaction (qRT-PCR). Receiver operating characteristic (ROC) analysis was utilized for evaluating the diagnostic value. Pearson's correlation analysis was used to analyze the correlation between A2M-AS1 and conventional AMI biomarkers. AMI-associated risk indicators were identified using logistic regression analysis.

Results A significant reduction of serum A2M-AS1 was measured in AMI patients relative to chest pain patients. A2M-AS1 had an area under the curve (AUC) of 0.927 to distinguish AMI patients from those with chest pain. Pearson's correlation analysis showed that A2M-AS1 was adversely correlated with white blood cell (WBC) (r=-0.6682, $P<0.001$), low density lipoprotein cholesterol (LDL-C) (r=-0.5795, $P<0.001$), creatine kinase MB (CK-MB) (r=-0.6022, $P<0.001$) and cTnI (r=-0.5473; $P<0.001$), while positively correlated with high density lipoprotein cholesterol (HDL-C) ($r=0.6445$, $P<0.001$). Relative to non-Major Adverse Cardiovascular Events (non-MACE) group, serum A2M-AS1 was obviously declined in the MACE group of AMI patients with high capacity to distinguish the MACE group from the non-MACE patients (AUC = 0.802). Additionally, A2M-AS1 ($P=0.013$; OR = 0.268; 95%CI = 0.095–0.760) was a risk indicator for predicting MACE with AMI patients, as well as age ($P=0.014$; OR = 3.478; 95%CI = 1.285–9.414).

Conclusion A reduction in A2M-AS1 expression was observed in AMI patients, suggesting its potential as an underlying indicator for AMI diagnosis.

Keywords LncRNA A2M-AS1, Diagnosis, MACE, AMI, ROC

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Introduction

Acute myocardial infarction (AMI) is a ubiquitous cardiovascular disorder, inducing ischemic necrosis of cardiomyocytes, with the inferior clinical outcome [1, 2]. The incidence of AMI has been increasing over the years threating the physical and mental health of human beings, which includes ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) [3]. At present, percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG), combined with thrombolytic therapy is the standard treatment for AMI [4]. The accurate and prompt detection of AMI is crucial in selecting the most appropriate treatment strategy, because it could prevent the progressive harmful damage to the myocardium and obtain favorable prognosis. Furthermore, the most pivotal aspect of diagnosing AMI is the evaluation of clinical symptoms and electrocardiographic (ECG) data, as well as conventional biomarkers [5]. The current diagnostic gold standard for AMI is the measurement of cardiac troponin (cTns) and creatinine kinase MB (CK-MB), following the recommendations set forth in the most recent clinical guidelines [6]. Nevertheless, cTns and CK-MB levels may be increased by other forms of cardiovascular pathology. So, exploration of novel and valuable predictors for screening AMI, particular for the early stage, is imminent.

Long non-coding RNAs (lncRNAs) comprising a length of over 200 nucleotides have been demonstrated to exert a pivotal influence on gene expression through a multitude of mechanisms, including transcriptional regulation, protein translation, and epigenetic inheritance [7]. LncRNAs are widely expressed in human tissues and have been shown to play a significant role in the pathological and cellular behaviors associated with a range of diseases [8]. For instance, Cao and others revealed that lncRNA-RMRP was markedly overexpressed in tissue samples of bladder cancer. This was found to facilitate bladder cancer cell proliferation, migration and invasion of bladder cancer cells [9]. Moreover, alterations in circulating lncRNA levels have been observed in the context of development-correlated diseases, such as cardiovascular system. Therefore, the use of lncRNA as a detection or prognostic biomarker is feasible. LncRNA-NRF was identified as a promising indictor for the screening of AMI with high accuracy [10]. As a promising lncRNA, alpha-2-macroglobulin-antisense 1 (A2M-AS1) has been reported to be aberrantly expressed in human tumors and cardiovascular diseases [11–13]. Nevertheless, it remains unclear whether A2M-AS1 can be regarded a valuable index for AMI in screening and predicting the clinical outcomes.

The present experiment examined A2M-AS1 expression levels in AMI patients and chest pain patients, and conducted a correlation analysis with conventional biomarkers. Furthermore, the potential clinical significance of A2M-AS1 levels in the early diagnosis and prognosis of patients attacked by AMI was also investigated.

Method and materials

Participants

A total of 100 AMI patients admitted to Shanghai Baoshan Luodian Hospital, diagnosed following WHO criteria, were recruited as the study group. At the same period, 80 age- and gender- matched chest pain (within 4 h) patients not suffering from cardiovascular or other organ-associated diseases served as the control group. Patients in the control group had normal coronary angiography and myocardial perfusion imaging without ischemia. Cardiac neuropathy, bone or muscle nerve inflammation are the most common causes of chest pain for the patients in the control group. All the subjects received a complete medical history, physical examination, and laboratory tests (WBC = white blood cell, TG = triglyceride, TC = total cholesterol, HDL-C = high density lipoprotein cholesterol, LDL-C = low density lipoprotein cholesterol, cTnI = cardiac troponin I, CK-MB).

Inclusion criteria were listed as follows: (1) AMI diagnosis in accordance with 2019 ESC (European Society of Cardiology) guidelines [14], (2) elevation of traditional cardiac biomarkers more than the upper limit of normal, (3) abnormal findings on ECG. Exclusion criteria were summarized as follows: (1) patients receiving anticoagulation or thrombolytic therapy; (2) with other late or serious diseases, such as malignancies, or organ failure.

Prior to the administration of treatments, 5 ml blood samples were collected from both the AMI group and the control group in the plain tubes containing EDTA anticoagulant. Serum was obtained by centrifugation at 4000 rpm for 10 min, and then rapidly preserved at -80℃ for the next experiments.

The study was conducted in accordance with the ethical standards set forth by the Ethics Committee of Shanghai Baoshan Luodian Hospital. All the participants provided the written informed consent for the application of their clinical information and biological specimens.

RNA isolation and quantitative real-time polymerase chain reaction (qRT-PCR)

Total RNA was extracted from serum samples using the RNeasy mini kit, following the protocols outlined by the manufacturer. The purified RNA was reverse-transcribed into cDNA with Reverse Transcription Kits (Thermo Fisher Scientific Inc., USA) based on the direction provided by the manufacturer. The relative quantification of A2M-AS1 was measured using Maxima SYBR Green qPCR Master Mix (Thermo-Fisher, USA). The housekeeping GAPDH was selected as the housekeeping gene

for normalization. The equation of $2^{-\Delta\Delta Ct}$ was applied to ascertain the relative amounts of A2M-AS1. Each experiment was conducted a minimum of three times.

Follow-up analysis

The patients with AMI were followed up for a period of six months following the administration of standard treatments. Based on whether MACE (Major Adverse Cardiovascular Events) occurred or not, all AMI patients were stratified into two groups: the non-MACE group and the MACE group.

Statistical analysis

SPSS 23.0 version and GraphPad Prism 7.0 software were adopted to statistically analyze the dada. Qualitative variables were represented as numbers (n) and percentages (%), and the differences between two groups were analyzed using Chi-square test. The continuous variables were reported as Mean ±SD (standard deviation), and a Student's t test was employed to assess the differences between two groups. Pearson's correlation analysis was conducted determined the relationship between A2M-AS1 and other biomarkers. The diagnostic efficacy of serum A2M-AS1 was assessed using the utilization of receiver operating characteristic (ROC) curves. A logistic regression analysis was conducted to ascertain the risk indicators of AMI patients. A *P* less than 0.05 was taken as a statistical significance.

Results

General information of all subjects

Table 1 showed the general information between the AMI patients and the control group. There was no statistical difference in age, sex, BMI (body mass index), diabetes, TG, and TC between two groups. There were 62 cases (62.0%) with smoking in the AMI group and 21 (26.3%) cases in the control group. There are 54 (54.0%) cases with drinking in the AMI group and 30 (37.5%) cases with drinking in the control group. In AMI patients, hypertension was present in 41 (41.0%) cases and 18 (22.5%) in the control group. A statistical difference was found in smoking, drinking and hypertension between two groups ($P<0.05$). What's more, the levels of HDL-C and WBC were significantly reduced in the AMI group in comparison to the control group ($P<0.001$). Significantly enforced levels in LDL-C, cTnI, CK-MB were observed in AMI patients relative to the control group (all $P<0.001$).

Table 1 Comparisons of clinical data between the two groups

Characteristics	Controls ($n=80$)	AMI ($n=100$)	*P*
Age (years)	59.69 ± 7.67	60.93 ± 7.55	0.311
Sex (male/female)	46/34	60/40	0.762
BMI (kg/m^2)	23.40 ± 2.58	23.73 ± 2.38	0.559
Smoking (n, %)	21 (26.3%)	62 (62.0%)	< 0.001
Drinking (n, %)	30 (37.5%)	54 (54.0%)	0.035
Hypertension (n, %)	18 (22.5%)	41 (41.0%)	0.011
Diabetes (n, %)	12 (15.0%)	26 (26.0%)	0.098
WBC (10^9/L)	6.99 ± 2.12	10.59 ± 2.44	< 0.001
TG (mmol/L)	1.78 ± 0.68	1.96 ± 0.75	0.104
TC (mmol/L)	4.77 ± 1.13	4.82 ± 1.04	0.761
HDL-C (mmol/L)	1.30 ± 0.34	1.06 ± 0.27	< 0.001
LDL-C (mmol/L)	2.46 ± 0.54	2.90 ± 0.66	< 0.001
cTnI (μg/L)	0.03 ± 0.02	1.59 ± 0.23	< 0.001
CK-MB (U/L)	15.30 ± 5.02	96.81 ± 11.94	< 0.001

Abbreviations: AMI, acute myocardial infarction; BMI, body mass index; WBC, white blood cell; TG, triglyceride; TC, total cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; BUN, blood urea nitrogen; cTnI, cardiac troponin I; CK-MB, creatine kinase MB. $P<0.05$ indicates significant different

Relative amounts of A2M-AS1 in AMI patients

To investigate the function of A2M-AS1 in the development of AMI, serum A2M-AS1 expression was measured in AMI patients and the control group. Results reveled that an obvious reduction of serum A2M-AS1 was observed in AMI patients compared with controls ($P<0.001$, Fig. 1A). More importantly, ROC curve revealed that the area under the curve (AUC) of serum A2M-AS1 was 0.927 to distinguish AMI patients from patients with chest pain with a sensitivity of 93.8% and a specificity of 83.0% (Fig. 1B).

Correlation analysis of A2M-AS1 with conventional biomarker of AMI

Pearson's correlation analysis was employed to ascertain the correlations of A2M-AS1 with WBC, HDL-C, LDL-C, CK-MB and cTnI (Fig. 2). A negative correlation was identified between serum A2M-AS1 and WBC (r=-0.6682, $P<0.001$, Fig. 2A), as well as LDL-C (r=-0.5795, $P<0.001$, Fig. 2C). Figure 2D demonstrated a negative correlation between serum A2M-AS1 and CK-MB (r=-0.6022, $P<0.001$,), while Fig. 2E illustrated a similar correlation between serum A2M-AS1 and cTnI (r=-0.5473; $P<0.001$). Conversely, serum A2M-AS1 was positively associated with HDL-C ($r=0.6445$; $P<0.001$, Fig. 2B).

Follow-up analysis

Following a six-month follow-up period, AMI sufferers were stratified into two groups: the non-MACE group and the MACE group, based on the occurrence of MACE. Relative to the non-MACE group, there was a notable decline in serum A2M-AS1 levels in the MACE group ($P<0.01$, Fig. 3A). In addition, serum A2M-AS1 exhibited a notable capacity to discriminate MACE from non-MACE. The AUC of serum A2M-AS1 was 0.802, with a sensitivity of 66.2% and a specificity of 86.2% (Fig. 3B).

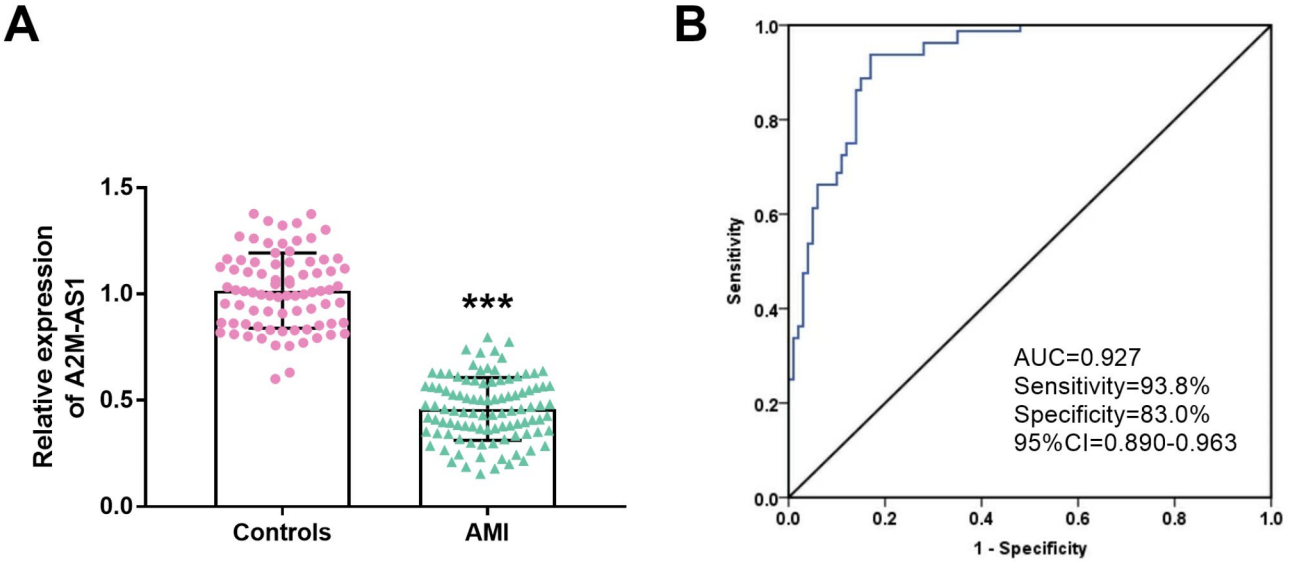

Fig. 1 Relative expression of A2M-AS1 in AMI was presented. **A**. A2M-AS1 was substantially diminished in AMI in comparison to the control group. **B**. A2M-AS1 demonstrated high diagnostic efficacy, with an AUC of 0.927. The sensitivity was 93.8%, and the specificity was 83.0%. (AMI: acute myocardial infarction; A2M-AS1: alpha-2-macroglobulin-antisense 1; AUC: the area under curves; ***: $P < 0.001$.)

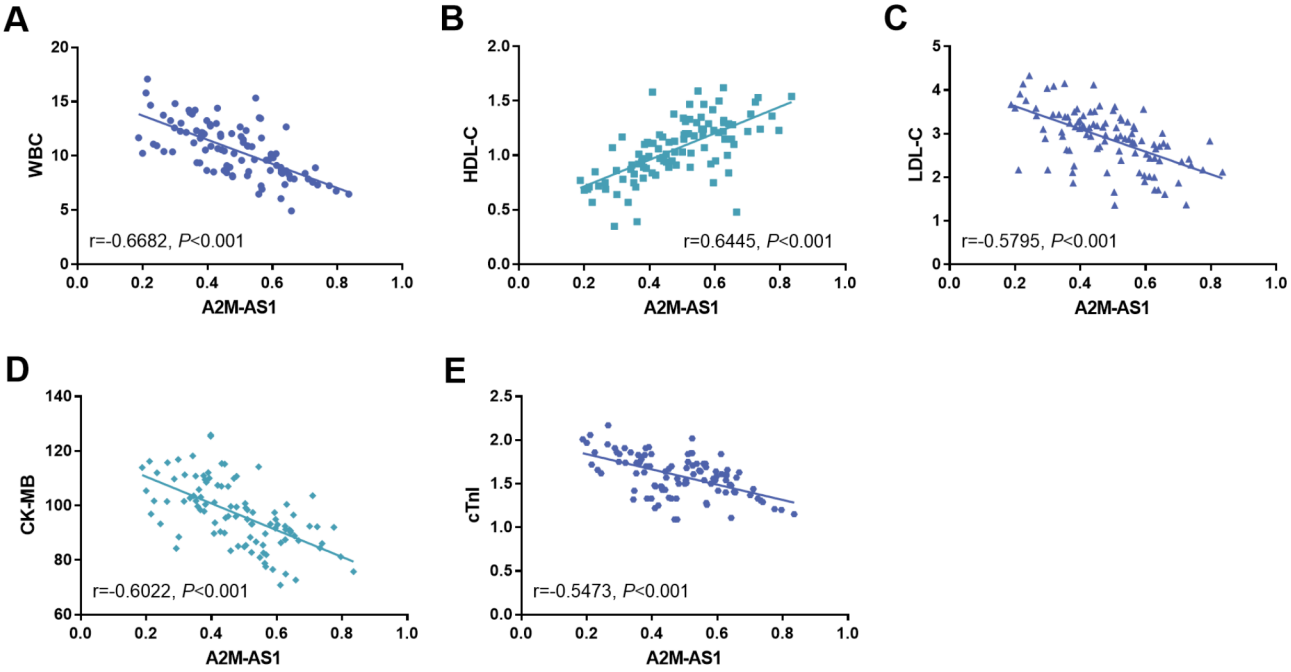

Fig. 2 Pearson's correlation was conducted to analyze the relationship of A2M-AS1 with the following variables: WBC (**A**), HDL-C(**B**), LDL-C (**C**), CK-MB (**D**) and cTnI (**E**). (A2M-AS1: alpha-2-macroglobulin-antisense 1; WBC: white blood cell; HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; CK-MB: creatine kinase MB; cTnI: cardiac troponin I.)

Univariate logistic regression of clinicopathological factors in MACE of AMI

In order to identify the risk factors associated with the occurrence of MACE in patients with AMI, univariate logistic regression was conducted. The independent variables included age, sex, BMI, smoking, drinking, hypertension, diabetes, WBC, TG, TC, HDL-C, LDL-C, cTnI, CK-MB and A2M-AS1.The dependent variable was whether AMI patients experienced MACE occurrence (Fig. 4). Results demonstrated that A2M-AS1 (OR = 0.220; 95%CI = 0.083–0.581; $P = 0.002$) served as a protective indicator of MACE. Age (OR = 3.621; 95%CI = 1.442–9.097; $P = 0.006$), hypertension (OR = 2.774; 95%CI = 1.142–6.740; $P = 0.024$) and cTnI

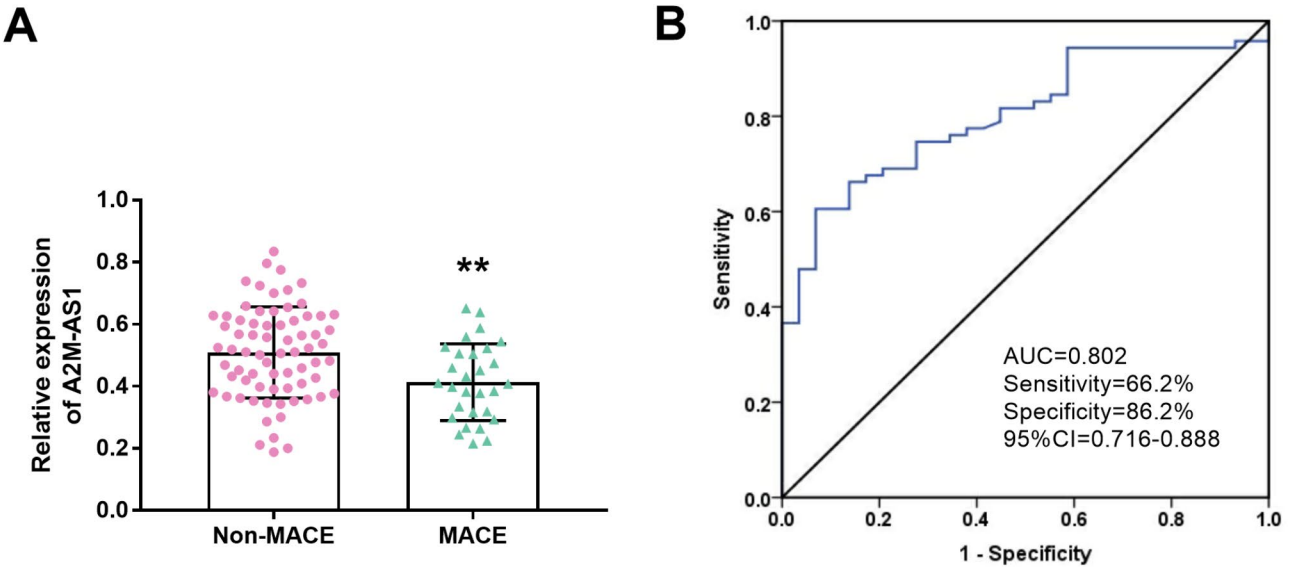

Fig. 3 Relative amounts of A2M-AS1 were examined in non-MACE group and MACE group of AMI patients. **A**: A2M-AS1 was notably decreased in the MACE group relative to the non-MACE group. **B**: The AUC of A2M-AS1 in discriminating between MACE from non-MACE groups was 0.802 with a sensitivity of 66.2% and a specificity of 86.2%. (MACE, major adverse cardiovascular events; A2M-AS1: alpha-2-macroglobulin-antisense 1; AUC: the area under curves; ***: $P < 0.001$.)

(OR = 2.752; 95%CI = 1.119–6.769; $P = 0.028$) were the risk factors of MACE (Table 2).

Multivariable logistic regression analysis of MACE in AMI

The statistically significant risk factors (age, hypertension, cTnI, A2M-AS1) identified in the univariate logistic regression analysis were considered as independent variables, with the occurrence of MACE in AMI patients taken as the dependent variable (Fig. 5). The results of multivariable logistic regression analysis indicated that A2M-AS1 (OR = 0.268; 95%CI = 0.095–0.760; $P = 0.013$) was an independently protective index for MACE, while age (OR = 3.478; 95%CI = 1.285–9.414; $P = 0.014$) was identified as an independent risk index (Table 3).

Table 2 Univariate logistics regression analyzed the risk factors of MACE in AMI patients

Independent variable	OR	95%CI		*P*
		upper limit	lower limit	
Age (years)	3.621	1.442	9.097	0.006
Sex (male/female)	1.130	0.465	2.743	0.787
BMI (kg/m^2)	1.727	0.720	4.139	0.221
Smoking (n, %)	1.921	0.750	4.920	0.174
Drinking (n, %)	1.591	0.658	3.846	0.303
Hypertension (n, %)	2.774	1.142	6.740	0.024
Diabetes (n, %)	0.871	0.320	2.366	0.786
WBC (10^9/L)	1.828	0.762	4.386	0.177
TG (mmol/L)	1.936	0.806	4.650	0.139
TC (mmol/L)	0.879	0.370	2.089	0.770
HDL-C (mmol/L)	0.791	0.332	1.880	0.594
LDL-C (mmol/L)	1.936	0.806	4.650	0.139
cTnI (μg/L)	2.752	1.119	6.769	0.028
CK-MB (U/L)	1.197	0.503	2.848	0.685
A2M-AS1	0.220	0.083	0.581	0.002

Abbreviations: MACE, major adverse cardiovascular events; AMI, acute myocardial infarction; BMI, body mass index; WBC, white blood cell; TG, triglyceride; TC, total cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; cTnI, cardiac troponin I; CK-MB, creatine kinase MB. $P < 0.05$ indicates significant different

Discussion

The majority of AMI is caused by the rupture or erosion of unstable plaque of coronary atherosclerosis, which is considered as the pathological basis of most acute coronary syndrome (ACS) [15]. Currently, a substantial number of biomarkers are employed for the detection, identification and diagnosis of acute cardiovascular events [16]. Early biochemical indicators, predominantly comprising cardiac enzyme markers and protein markers, are serve as a crucial foundation for AMI diagnosis in clinical settings. Enzyme markers include lactate dehydrogenase (LDH), aspartate aminotransferase (AST), α-hydroxybutyrate dehydrogenase, CK, CK-MB, and so on [17]. Protein makers contain myoglobin and cTn [18]. Nevertheless, it has been reported that some biomarkers are unreliable due to the occurrence of numerous false positive or false negative outcomes. It is therefore imperative that patients exhibiting suggestive symptoms of an AMI are promptly and accurately diagnosed and treated.

It has been reported that, because of their high stability in blood and low susceptibility to nucleases degradation by, lncRNAs are more reliable than other circulating nucleic acids [19]. The alteration of circulating lncRNA

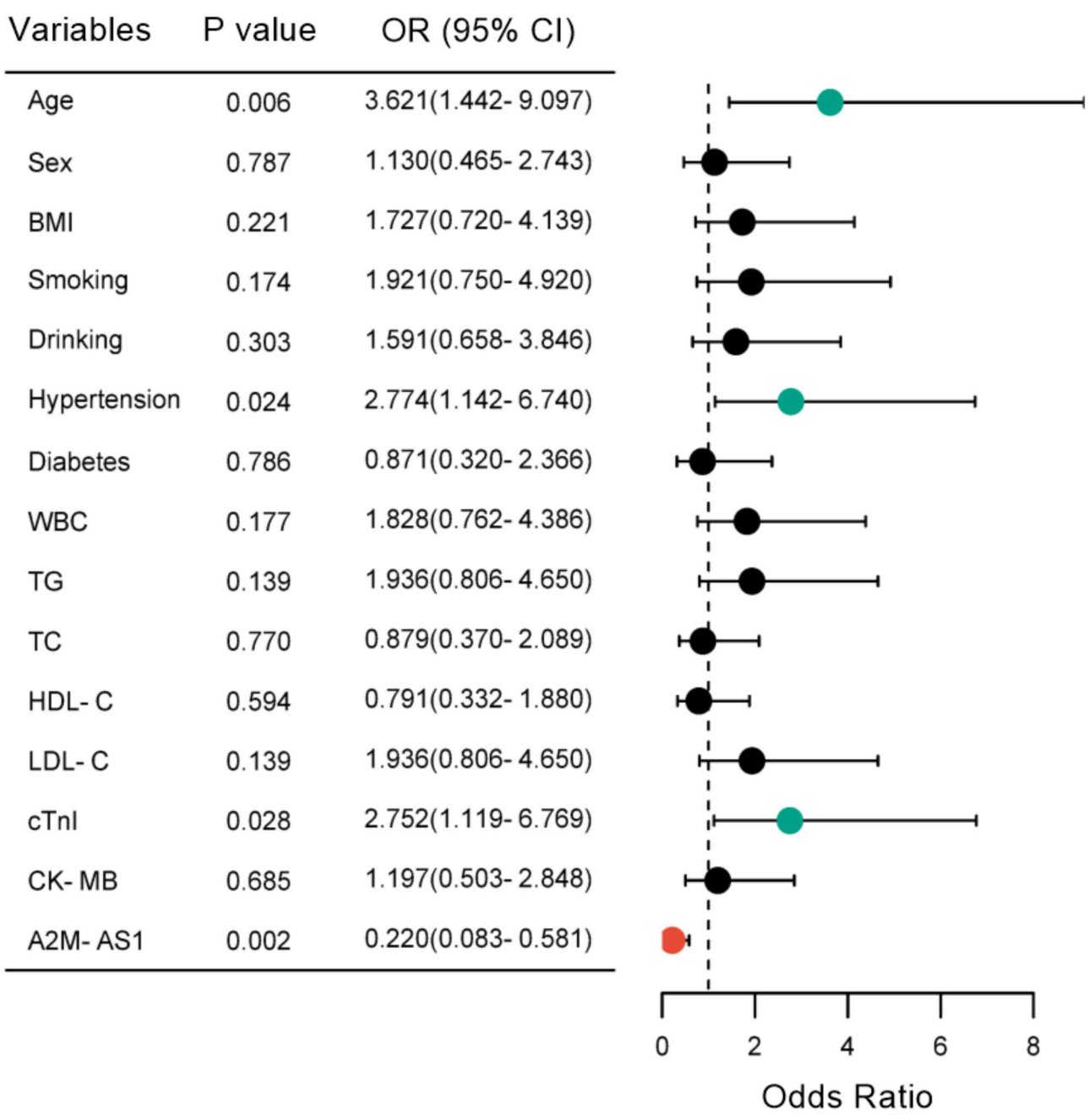

Fig. 4 Univariable logistic regression analysis was conducted for occurrence of MACE. (MACE: major adverse cardiovascular events.)

levels is particularly implicated in the progression of diseases. Jin et al. demonstrated that lncRNA DRAIR was widely reported in gastric cancer tissues and exhibited a positive correlation with its expression in plasma. The up-regulation of DRAIR may serve as an underlying indicator for the detection and progression of gastric cancer [20]. An increasing body of evidence substantiates the efficacy of lncRNAs in the diagnosis of AMI with the assertion that lncRNAs acted a pivotal role in the initiation and formation of AMI. For instance, Xie et al. manifested that lncRNA TTTY15 was upregulated in AMI patients, while lncRNA HULC was down-regulated. The combination of TTTY15 and HULC has been demonstrated to possess a high diagnostic potency for the prediction AMI occurrence [21]. Azat and others have indicated that the levels of lncRNA MIAT were markedly elevated in patients with AMI, thus representing a promising detection indicator. The silencing of MIAT was observed to notably suppress cardiomyocyte apoptosis [22].

The preliminary findings of this study indicate a significant reduction in A2M-AS1 levels in patients with AMI,

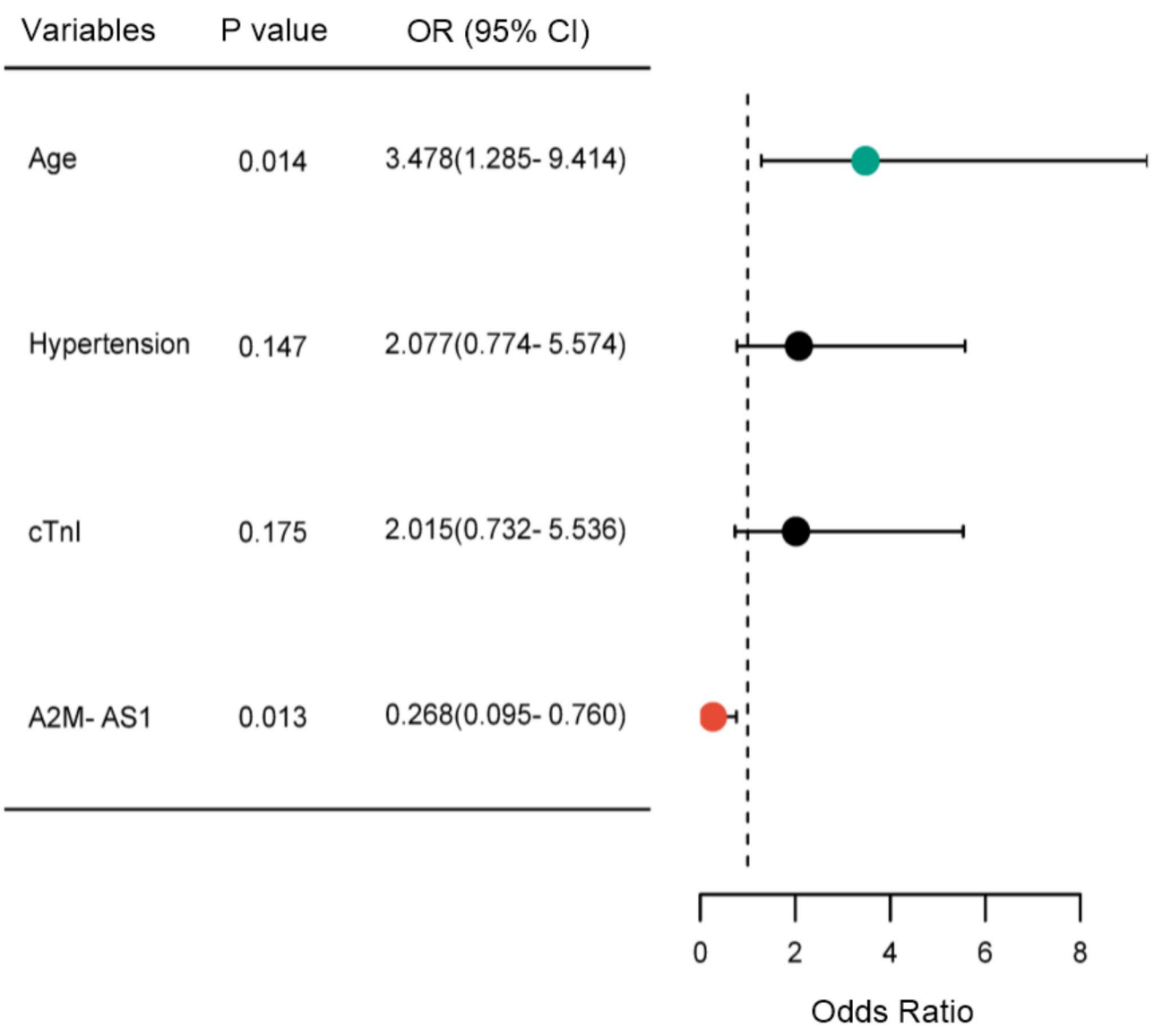

Fig. 5 Multivariable logistic regression analysis was conducted for occurrence of MACE. (MACE: major adverse cardiovascular events.)

which may facilitate the early diagnosis of AMI. Furthermore, A2M-AS1 demonstrated the capacity to effectively discriminate AMI patients who experienced MACE with a high degree of accuracy. The results demonstrated that A2M-AS1 was an independent risk factor for predicting MACE, as well as age in AMI patients. These findings were consistent with those previously reported in the literature. Li and others asserted that A2M-AS1 was substantially diminished in AMI patients based on two microarray datasets, suggesting its potential as a potential diagnostic biomarker for AMI [11]. Song et al. illustrated that A2M-AS1 levels were markedly reduced in cardiomyocytes subjected to hypoxia/reoxygenation. A high level of A2M-AS1 has been demonstrated to significantly facilitate cell growth of cardiomyocytes by down-regulating interleukin 1 receptor type 2 (IL1R2) [12]. Yu and others demonstrated that A2M-AS1 expression was significantly diminished in AMI patients and hypoxia/reperfusion-treated cardiomyocytes via the miR-556-5p/XIAP (X-linked inhibitor of apoptosis protein) axis [23].

Table 3 Multivariate logistics regression analyzed the risk factors of MACE in AMI patients

Independent variable	OR	95%CI		*P*
		upper limit	lower limit	
Age (years)	3.478	1.285	9.414	0.014
Hypertension (n, %)	2.077	0.774	5.574	0.147
cTnI (μg/L)	2.015	0.732	5.536	0.175
A2M-AS1	0.268	0.095	0.760	0.013

Abbreviations: MACE, major adverse cardiovascular events; AMI, acute myocardial infarction; cTnI, cardiac troponin I. $P<0.05$ indicates significant different

Furthermore, the present study revealed a negative correlation between A2M-AS1 levels and WBC, LDL-C, CK-MB, and cTnI in patients who had experienced an AMI. Conversely, a positive correlation was identified between A2M-AS1 expression and HDL-C. The levels of A2M-AS1 may be regarded to be a relevant index for the early detection of AMI and have a certain predictive effect on the prognosis of AMI. This may assist clinicians in better judging the occurrence, severity and prognosis of AMI to a certain extent. In pancreatic ductal adenocarcinoma, A2M-AS1 has been identified as a novel prognostic indicator and therapeutic target through the use of weighted gene co-expression network analysis [24]. In lung adenocarcinoma, A2M-AS1 was identified as a potential biomarker through bioinformatics analysis [25]. Zhou et al. demonstrated that A2M-AS1 was aberrantly expressed in non-small cell lung cancer and plays a role in the development of the tumor, establishing it as a new indicator for non-small cell lung cancer [26].

Oxidative stress and apoptosis have been proved to be the underlying mechanisms of aberrant A2M-AS1 in cardiomyocytes [23]. Firstly, oxidative stress can directly damage important molecules of cells, such as DNA and proteins, leading to initiation of apoptosis. Moreover, oxidative stress could influence the expression and activities of apoptosis-related proteins via regulating the signaling pathway. The relationship between oxidative stress and apoptosis is intricate and intertwined, both of which influence the development and progression of cardiomyocytes.

There are several limitations which should be explained in our research. First of all, the follow-up time is short, and the sample size is insufficient. Then, there was bias. In the future research, multi-center, randomized and double-blind research can be adopted. The sample size can be expanded and the follow-up time should be increased, and the alteration of A2M-AS1 level can be monitored dynamically, namely tracking patients over time so as to assess the predictive value of A2M-AS1 for future cardiac events. In the future, further research is required to investigate whether the increase of A2M-AS1 level can improve the clinical outcome of AMI patients.

Conclusion

A2M-AS1 was found to be markedly reduced in AMI patients and may serve as a promising indicator for the early detection of AMI. A reduction in A2M-AS1 expression may serve to discriminate MACE from non-MCAE in AMI patients, with a high degree of diagnostic capacity.

Abbreviations

A2M-AS1	alpha-2-macroglobulin-antisense 1
AMI	Acute myocardial infarction
AST	Aspartate aminotransferase
AUC	Area under the curve
BMI	Body mass index
CABG	Coronary artery bypass graft
CK-MB	Creatinine kinase MB
cTnI	Cardiac troponin I
ECG	Electrocardiographic
HDL-C	High density lipoprotein cholesterol
IL1R2	Interleukin 1 receptor type 2
LDH	Lactate dehydrogenase
LDL-C	Low density lipoprotein cholesterol
LncRNA	Long non-coding RNA
NSTEMI	Non-ST-segment elevation myocardial infarction
PCI	Percutaneous coronary intervention
qRT-PCR	Quantitative real time polymerase chain reaction
ROC	Receiver operating characteristic
STEMI	ST-segment elevation myocardial infarction
TC	Total cholesterol
TG	Triglyceride
WBC	White blood cell

Acknowledgements

Not Applicable.

Author contributions

Author XY H, author HL J, author BQ Z and author LJ Z have given substantial contributions to the conception or the design of the manuscript, author B X, author J C and author YJ S to acquisition, analysis and interpretation of the data. CM C and QY H wrote the manuscript. All authors have participated to drafting the manuscript and approved the final version of the manuscript.

Funding

This study was supported by Nomogram analysis of serum hsCRP, apoB/apoA1 levels and related risk factors in patients with acute coronary syndromes(CSFGG-2022166).

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Shanghai Baoshan Luodian Hospital before the study began. The participants' right to be informed about the study was ensured and agreed to participate in the study.

Consent for publication

All patients provided written informed consent.

Competing interests

The authors declare no competing interests.

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Received: 20 September 2024 / Accepted: 9 March 2025
Published online: 25 March 2025

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