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The inflammatory markers MHR and NLR are independent risk factors for adverse events during hospitalization in older adult patients with myocardial injury caused by acute carbon monoxide poisoning: a retrospective cross-sectional study

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Abstract

Acute carbon monoxide poisoning-induced myocardial injury is easily overlooked. However, patients with acute carbon monoxide poisoning, especially older adult patients, often have difficulty expressing clinical symptoms due to early consciousness disturbances, making the early identification of complications challenging and leading to delayed diagnosis and treatment. Therefore, exploring indicators that can predict in-hospital cardiovascular adverse events in older adult patients with acute carbon monoxide poisoning-induced myocardial injury has important clinical significance. Therefore, this retrospective cross-sectional study included older adult patients with acute carbon monoxide poisoning-induced myocardial injury at the Department of Hyperbaric Oxygen of Beijing Chao-Yang Hospital from January 2013 to December 2019. A total of 119 older adult patients with acute carbon monoxide poisoning-induced myocardial injury were included in the study, with 94 patients in the nonevent group (54 males, 40 females, 71.09 ± 7.60 years) and 25 patients in the cardiovascular adverse event group (10 males, 15 females, 71.48 ± 10.38 years). Compared with those in the nonevent group, creatine kinase isoenzyme levels, triglyceride levels, the neutrophil/lymphocyte ratio and the monocyte/high-density lipoprotein cholesterol ratio were significantly greater in the cardiovascular adverse event group, and high-density lipoprotein cholesterol levels were significantly lower in the cardiovascular adverse event group. Further binary logistic regression analysis showed that higher monocyte/high-density lipoprotein cholesterol ratio might be an independent risk factor for in-hospital adverse events in older adult patients with acute carbon monoxide poisoning-induced myocardial injury (*OR* = 109.783, 95% *CI*: 2.644–4557.834; *P* = 0.013). The area under the curve of the monocyte/high-density lipoprotein cholesterol ratio in predicting in-hospital cardiovascular adverse events in older adult patients with myocardial injury due to acute carbon monoxide poisoning was 0.797, the cutoff value was 0.645, the sensitivity was 68.0%, and the specificity was 88.2%. The inflammatory indicators monocyte/high-density lipoprotein cholesterol ratio and neutrophil/lymphocyte ratio were identified as an independent risk factor for predicting in-hospital cardiovascular adverse events in older adult patients with acute carbon monoxide poisoning-induced myocardial injury in this study. Specifically, monocyte/high-density lipoprotein cholesterol ratio was identified as an independent risk factor for predicting adverse events during hospitalization.

Key Words: acute carbon monoxide poisoning; binary logistic regression analysis; creatine kinase isoenzyme MB; elderly; hyperbaric oxygen treatment; in-hospital cardiovascular adverse events; monocyte/high-density lipoprotein cholesterol ratio; myocardial injury; neutrophil/lymphocyte ratio; predictive value

Introduction

Carbon monoxide (CO) is the most common cause of fatal poisoning worldwide because it is colorless, odorless, and nonirritating.[1] CO poisoning (COP) is a significant public health issue worldwide, with particular prominence in developing countries.

CO has a strong affinity for human hemoglobin. Carboxyhemoglobin formed during COP reduces the delivery of oxygen to tissues, leading to hypoxic damage to vital organs.[2] The heart, which requires the most oxygen, is therefore most susceptible to injury. Myocardial injury is a complication of acute COP (ACOP), with an incidence

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rate of approximately 20–37% and a poor prognosis.[3,4] After ACOP occurs, CO enters the human body and forms a large amount of carboxyhemoglobin, reducing the myocardial oxygen reserve and leading to metabolic disorders in myocardial cells and anaerobic glycolysis, which causes lactic acid accumulation and lipid deposition and induces myocardial cell injury.[5] Moreover, a variety of factors lead to a systemic inflammatory response, and inflammation causes widespread endothelial dysfunction and further suppression of heart function.[6] Additionally, CO stimulates the sympathetic nerve to produce a large amount of catecholamines, causing vasoconstriction and an increased heart rate, aggravating the burden on the heart and leading to a decrease in cardiac function.[7] Moreover, the destruction of myocardial cell oxidative phosphorylation leads to a reduction in adenosine triphosphate, as well as changes in calcium gradients affecting action potentials, leading to prolonged repolarization, thereby affecting the conduction function of myocardial cells.[8] During this process, patients may experience various cardiovascular events, including atrial fibrillation, ventricular premature beats, sinus tachycardia, myocardial infarction, and even death.[9]

ACOP with myocardial injury often occurs covertly and is easily overlooked because of its reversible characteristics.[10] As a special group of poisoning victims, the elderly have more underlying diseases, and the impact of hypoxia on them is more severe. They are more difficult to treat, have a poorer prognosis, and often suffer from serious cardiovascular events such as severe arrhythmias, myocardial infarction, acute heart failure, and even sudden death.[9]

Elderly patients not only have a natural decline in cardiac function but also in other organs and have poor tolerance to diseases, making them more prone to adverse outcomes from COP. The duration of coma reflects the severity of COP. During coma, patients may suffer from continuous hypoxia and metabolic disorders, which can lead to multiorgan dysfunction, especially more severe damage to the heart and brain. Timely HBOT can reduce the duration of coma from COP, thereby improving the prognosis of the heart and brain.[11]

Oxygen therapy is the preferred treatment for COP. The half-life of CO is 5 hours with spontaneous breathing in room air; it is 1.5 hours with 100% oxygen; and it is 25 minutes with hyperbaric oxygen therapy (HBOT).[12] HBOT is recommended for patients with neurological symptoms and myocardial involvement.[13]

However, patients with ACOP, especially older adult patients, often have difficulty expressing clinical symptoms due to early consciousness disturbances, making the early identification of complications challenging and leading to delayed diagnosis and treatment. Therefore, exploring indicators that can predict in-hospital cardiovascular adverse events in elderly patients with ACOP and myocardial injury has important clinical significance.

Inflammatory responses are closely related to ACOP. Recently, the monocyte/high-density lipoprotein cholesterol ratio (MHR) and neutrophil/lymphocyte ratio (NLR) have emerged as simple and easily obtained novel inflammatory indicators in clinical practice and are widely applied in clinical inflammatory diseases.[14-17] They can well predict the prognosis of inflammation-related diseases (such as pneumonia) and the prognosis after cardiac arrest and hemorrhagic or ischemic stroke[18-20] and have predictive value for the occurrence of cardiovascular events.[21,22]

Therefore, this retrospective cross-sectional study uses indicators such as the MHR to predict in-hospital adverse cardiovascular events in elderly patients with ACOP myocardial injury, providing a basis for the early monitoring, treatment, and prognosis of elderly patients with ACOP myocardial injury.

Subjects and Methods

Subjects

Older adult patients with ACOP and myocardial injury treated in the Department of Hyperbaric Oxygen, Beijing Chao-Yang Hospital Affiliated to Capital Medical University from January 2013 to December 2019 were selected. The inclusion criteria were as follows: (1) aged ≥ 60 years[23]; (2) admitted within 24 hours of onset; (3) acute moderate to severe COP[24]; and (4) myocardial injury due to ACOP.[25] The exclusion criteria were as follows: (1) the presence of atrial fibrillation, ventricular tachycardia, atrioventricular block, paroxysmal supraventricular tachycardia, cardiogenic shock, ventricular fibrillation, or other cardiovascular adverse events at the time of presentation; (2) concurrent cardiomyopathy, coronary heart disease, or chronic heart failure; (3) concurrent chronic obstructive pulmonary disease, bronchial asthma, or pulmonary interstitial fibrosis; (4) concurrent severe infection upon admission; (5) concurrent systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, or other autoimmune diseases, or the use of immunosuppressive drugs within 3 months; (6) concurrent hematological diseases; and (7) concurrent malignant tumors. The study complies with the STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) (**Additional file 1**).[26] All appropriate patient consent forms were obtained.

Study grouping

Patients were divided into a nonevent group and an event group on the basis of whether they experienced in-hospital cardiovascular adverse events. In-hospital cardiovascular adverse events are defined as follows: (1) severe arrhythmias, including atrial fibrillation, atrioventricular block, paroxysmal supraventricular tachycardia, ventricular tachycardia, ventricular fibrillation, and severe sinus arrest; (2) angina and acute myocardial infarction; (3) acute heart failure; and (4) sudden cardiac death. Patients who experienced two or more events simultaneously or successively were not recorded repeatedly.

Treatment

All patients were given comprehensive treatment, including cardiac monitoring, oxygen therapy, HBOT, nutritional support for the myocardium, improvement of circulation, and prevention of cerebral edema.[27] HBOT consisted of at least four courses: Inhalation of 100% oxygen through an oral-nasal mask in a hyperbaric chamber at 2.0 atmosphere absolute, treatment time 120 minutes (compression 30 minutes—oxygen inhalation 60 minutes—decompression 30 minutes), each course consisting of 10 sessions (1 session/day, 6 sessions/week), with a 1-week pause after every two courses before continuing treatment.

Observational parameters

Data on patient sex, age, smoking and alcohol consumption, comorbidities (hypertension, diabetes, hyperlipidemia, and anemia), poisoning situations (transient loss of consciousness, and coma duration), and delayed encephalopathy manifestations (mental retardation, psychological disorders, muscle tension disorders, and epilepsy) were collected upon admission. Arterial blood was drawn for the detection of carboxyhemoglobin. Venous blood was drawn to test for complete blood count (including white blood cells, neutrophils, lymphocytes, monocytes, and platelets), biochemistry (including blood lipids: total cholesterol, high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol, triglycerides, NLR, and MHR, and cardiac enzymes (including creatine kinase (CK), creatine kinase isoenzyme (CK-MB), cardiac troponin I (cTNI), N-terminal pro-brain natriuretic peptide, and D-dimer). To observe whether patients presented other

complications, we monitored for the presence of cerebral edema, pulmonary edema, pulmonary infection, cerebral infarction, and damage to renal function.

Data collection

The project team conducted training and established standardized procedures for data collection, which were completed at the time of patient visits and corresponding intervention points. Patients were enrolled and managed according to the research protocol, with each patient's data being promptly and completely recorded on an individual form, ensuring accurate documentation. Regular data review was conducted to guarantee the authenticity and reliability of the research content, and one member of the project team was designated a quality control officer.

Sample size calculation

A medium effect size is assumed, represented by an odds ratio (OR) of 2.0, on the basis of previous studies. The significance level (α) is typically set at 0.05. The power ($1 - \beta$) was set at 0.80. The event rate (p), which is the prevalence of in-hospital cardiovascular adverse events in elderly patients with ACOP, was estimated to be 30%. The sample size (n) can be calculated using the following formula:

$$n = \left(\frac{Z_{1-\alpha/2}+Z_{1-\beta}}{\ln(\mathrm{OR})\times\sqrt{p(1-p)}}\right)^2.$$

The sample size is at least $n \approx 78$.

Statistical analysis

Data analysis was conducted using IBM SPSS Statistics for MacOS (Version 23.0 (IBM Corp, Armonk, NY, USA), with the Kolmogorov–Smirnov test used to determine if the data were normally distributed. The quantitative data are expressed as the mean ± standard deviation (SD), and group comparisons were made using the independent samples t-tests. Categorical data were analyzed using chi-square tests. Binary logistic regression analysis was used to identify risk factors for in-hospital cardiovascular adverse events. The predictive value of in-hospital cardiovascular adverse events in elderly patients with myocardial injury due to ACOP was evaluated using the receiver operating characteristic (ROC) curves. $P < 0.05$ was considered statistically significant.

Results

Comparison of basic information between the cardiovascular event and nonevent groups

A total of 94 patients without cardiovascular events (nonevent group) and 25 patients with cardiovascular events (event group) were included in this study. The two groups were compared in terms of age, sex, smoking history, drinking history, and comorbidities (including hypertension, diabetes, hyperlipidemia, and anemia). The results revealed that there were no significant differences in the baseline parameters between the two groups (all $P > 0.05$; **Table 1**).

Comparison of clinical characteristics between the cardiovascular event and nonevent groups

In terms of clinical characteristics, there were no significant differences in the proportion of transient loss of consciousness or duration of coma between the two groups (all $P > 0.05$). Delayed encephalopathy manifestations (including mental retardation, psychological disorders, muscle tension disorders, and epilepsy) and complications (including cerebral edema, pulmonary edema, pulmonary infection, cerebral infarction, and renal function damage) were not significantly different between the two groups (all $P > 0.05$; **Table 1**).

Table 1 | Demographic and clinical characteristics of the cardiovascular adverse event and nonevent groups

Parameter	Nonevent group (n = 94)	Event group (n = 25)	t/U value	P-value
Age (yr)	71.09±7.60	71.48±10.38	0.213	0.831
Sex (male:female)	54:40	10:15	970	0.175
Smoking [n(percentage)]	34(36)	7(28)	1079	0.488
Drinking [n(percentage)]	24(25)	4(16)	1063	0.430
Comorbidity [n(percentage)]				
Hypertension	42(45)	8(32)	1026	0.268
Diabetes	18(19)	8(32)	1024	0.181
Hyperlipidemia	24(26)	5(20)	1110	0.615
Anemia	6(6)	3(12)	1109	0.395
Poisoning situation				
Transient loss of consciousness [n(percentage)]	82(87)	25(100)	1025	0.069
Coma duration (h)	10.62±13.01	11.08±9.60	0.166	0.868
Delayed encephalopathy manifestation [n(percentage)]				
Mental retardation	26(28)	8(32)	1124	0.804
Psychological disorder	26(28)	10(40)	1030	0.327
Muscle tension disorder	8(8)	5(20)	1040	0.143
Epilepsy	2(2)	2(8)	1106	0.194
Complication [n(percentage)]				
Cerebral edema	12(13)	3(12)	1166	> 0.999
Pulmonary edema	4(4)	0	1125	0.578
Pulmonary infection	48(51)	12(48)	1139	0.825
Cerebral infarction	10(11)	5(20)	1065	0.305
Renal function damage	8(8)	0	1075	0.201

The quantitative data are expressed as the mean ± SD and were analyzed using t-tests. Categorical data were analyzed by chi-square tests.

Comparison of laboratory parameters between the cardiovascular event and nonevent groups

The level of CK-MB was significantly greater in the event group than in the nonevent group (P = 0.031). There were no significant differences in D-dimer, creatine kinase, cTNI, or N-terminal pro-brain natriuretic peptide between the two groups (all P > 0.05).

In terms of blood lipid levels, HDL was significantly lower in the event group than in the nonevent group (P = 0.010). The level of triglycerides was also greater in the event group than in the nonevent group (P = 0.036). Total cholesterol and low-density lipoprotein cholesterol did not significantly differ between the two groups (all P > 0.005).

The NLR and MHR were also significantly greater in the event group than in the nonevent group (P = 0.027 and P = 0.019; **Table 2**).

Binary logistic regression analysis

The results showed that a higher MHR might be an independent risk factor for in-hospital cardiovascular adverse events in elderly patients with ACOP myocardial injury (P = 0.013; **Table 3**).

Receiver operating characteristic curve analysis

Receiver operating characteristic curve analysis revealed that the area under the curve of the MHR in predicting in-hospital cardiovascular adverse events in elderly patients with myocardial injury due to ACOP was 0.797, the cutoff value was 0.645, the sensitivity was 68.0%, and the specificity was 88.2% (P = 0.000; **Figure 1**).

Table 2 | Laboratory parameters of the cardiovascular adverse event and nonevent groups

Parameters	Nonevent group (n = 94)	Event group (n = 25)	t/U value	P-value
COHB (%)	19.39±10.95	22.01±14.49	0.539	0.593
D-dimer (mg/L)	80.87±194.20	77.60±185.10	0.049	0.961
CK (U/L)	295.90±427.70	373.10±344.30	0.499	0.621
CK-MB (U/L)	1.13±1.22	2.46±2.34	2.248	0.031
cTNI (ng/L)	0.31±1.58	0.66±1.08	0.647	0.521
NT-proBNP (pg/mL)	1983±7575	4231±5510	0.762	0.453
CHOL (mM)	4.06±0.59	3.70±0.85	1.307	0.202
HDL (mM)	1.16±0.35	0.78±0.17	2.770	0.010
LDL (mM)	2.28±0.59	2.10±0.58	0.724	0.475
TG (mM)	0.94±0.53	1.37±0.74	2.149	0.036
WBC (10^9/L)	7.19±2.17	8.37±2.59	1.483	0.145
NE (10^9/L)	4.88±2.06	6.24±2.79	1.725	0.091
LY (10^9/L)	1.61±0.65	1.24±0.56	1.619	0.112
MO (10^9/L)	0.50±0.23	0.48±0.15	0.245	0.807
PLT (10^9/L)	193.1±63.95	241.8±130.10	1.687	0.099
NLR	3.80±2.88	6.58±5.13	2.282	0.027
MHR	0.43±0.18	0.65±0.25	2.502	0.019

The quantitative data are expressed as the mean ± SD and were analyzed using t-tests. CHOL: Total cholesterol; CK: creatine kinase; CK-MB: creatine kinase isoenzyme MB; COHB: carboxyhemoglobin; cTNI: cardiac troponin I; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein cholesterol; LY: lymphocytes; MHR: monocyte-to-high-density lipoprotein cholesterol ratio; MO: monocytes; NE: neutrophils; NLR: neutrophil/lymphocyte ratio; NT-proBNP: N-terminal pro-B-type natriuretic peptide; PLT: platelet; TG: triglycerides; WBC: white blood cells.

Table 3 | Binary logistic regression analysis of independent risk factors for adverse in-hospital events in older adult patients with ACOP myocardial injury

Factor	B-value	SE	Wald	P-value	$Exp(B)$	95% CI
NLR	0.183	0.137	1.778	0.182	1.201	0.918–1.571
MHR	4.699	1.901	6.108	0.013	109.783	2.644–4557.834
CK-MB	0.160	0.207	0.594	0.441	1.173	0.782–1.761
Constants	–4.599	1.446	10.116	0.001	0.010	

ACOP: Acute carbon monoxide poisoning; CI: confidence interval; CK-MB: creatine kinase isoenzyme MB; MHR: monocyte-to-high-density lipoprotein cholesterol ratio; NLR: neutrophil/lymphocyte ratio.

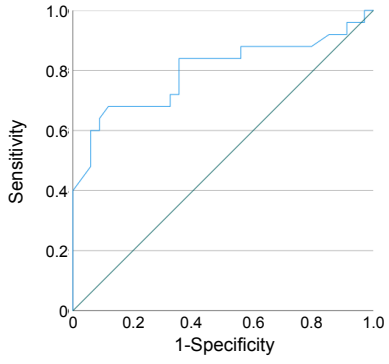

Figure 1 | Receiver operating characteristic curve of the MHR for predicting in-hospital cardiovascular adverse events in older adult patients with myocardial injury due to ACOP.
ACOP: Acute carbon monoxide poisoning; MHR: monocyte-to-high-density lipoprotein cholesterol ratio.

Discussion

cTNI and CK-MB are two recognized markers of cardiac injury, and their concentrations can reflect the degree of myocardial cell injury.[28,29] Baydin et al.[30] found that ACOP patients with myocardial injury had higher serum levels of cTNI and CK-MB than those without myocardial injury. The results of this study revealed no difference in cTNI levels between the two groups. Although there was a statistically significant difference in CK-MB levels, it was not a predictive factor for in-hospital adverse cardiovascular events in the regression analysis. The reason for this may be that most ACOP patients are admitted with impaired consciousness, making it difficult to determine the exact onset time of the disease, which prevents the measurement of accurate peak values for cTNI and CK-MB. On the other hand, although the two parameters were not statistically significant, the values in the event group were still greater than those in the nonevent group, and the differences in the data may be statistically significant with increasing sample size. N-terminal pro-brain natriuretic peptide is secreted mainly by ventricular muscle cells and is used to assess cardiac function.[31] The lack of statistically significant differences in the results is also considered to be due to the aforementioned issues.

Dyslipidemia (especially increased triglycerides and decreased HDL) may be related to atherosclerosis, endothelial dysfunction, and inflammatory responses, all of which are risk factors for cardiovascular diseases.[32,33] In the context of COP, dyslipidemia may exacerbate damage to the heart and vascular system, thereby increasing the risk of adverse events.

Inflammatory responses play a significant role in ACOP.[34] Our results revealed that the NLR was significantly greater in the event group than in the nonevent group, which is consistent with the conclusions of other researchers.[34] This finding also reflects the adverse effects of imbalanced inflammatory regulation on cardiovascular events in older adult individuals. The NLR is an inflammatory marker that comprehensively evaluates neutrophils and lymphocytes, reflecting proinflammatory states. Compared with the single white blood cell count, the NLR is more stable and predictable. It is the ratio of two different yet complementary immune pathways, combining the activation of neutrophils in response to nonspecific inflammation and the dominant role of lymphocytes in regulating inflammation.[35] After ACOP, the body may be in a state of stress. Under stress, the levels of catecholamines and cortisol increase. Catecholamines can increase the release of neutrophils.[36] Cortisol can increase the content of neutrophils in peripheral blood and reduce the degree of neutrophil apoptosis.[35,37,38] A decrease in lymphocytes can reflect the intensity of stress events and the resistance and adaptability of the immune system, leading to a decline in cellular immunity.[39] High neutrophil counts cause severe ischemic damage and poor prognosis, whereas lymphocytes have anti-inflammatory and endothelial-protective effects, a reduction in lymphocytes is detrimental to controlling inflammation.[39] Moreover, catecholamines cause vasoconstriction and increased heart rate, aggravating the burden on the heart and leading to a decline in cardiac function. In patients with high NLRs, strict monitoring may help with early identification of the occurrence of in-hospital cardiovascular events in elderly ACOP patients and in making treatment decisions to prevent the occurrence of in-hospital cardiovascular events. As a simple and cost-effective indicator, the NLR may help quickly assess the inflammatory state of elderly patients and predict the risk of adverse events.

The MHR is a new indicator of systemic inflammation obtained by dividing the monocyte count in peripheral blood by the HDL value and is also considered a predictive factor for cardiovascular events. Monocytes participate in the release of proinflammatory and pro-oxidative cytokines at inflammatory sites and play important roles in chronic inflammation and cardiovascular diseases. Monocytes play a significant role in the release of proinflammatory and pro-oxidative cytokines at sites of inflammation, and they are important in chronic inflammation and cardiovascular diseases. They may trigger the formation, progression, and rupture of atherosclerosis and can predict the formation of new plaques, leading to plaque instability.[40] Conversely, HDL has anti-inflammatory and antioxidant effects, which are expressed by inhibiting the production and mobilization of monocytes, preventing the migration of macrophages to the arterial wall and removing cholesterol molecules from these cells.

Additionally, HDL possesses anti-inflammatory and fibrinolytic properties, which can protect the cardiovascular system and delay the progression of atherosclerosis. The MHR is more accurate than a single indicator because of the decrease in monocytes and the increase in HDL. HDL levels are inversely correlated with coronary heart disease.[41] Açıkgöz et al.[42] emphasized the role of the MHR as an independent predictive factor for major adverse cardiovascular events in patients with acute ST-segment elevation myocardial infarction. In this study, the MHR was significantly greater in the adverse events group than in the nonevents group, and at the same time, a higher MHR was an independent risk factor for in-hospital adverse cardiovascular events in elderly patients with ACOP myocardial injury. The MHR can be used as a practical, cost-effective, and highly predictive marker for cardiovascular adverse events in older adult patients with ACOP myocardial injury.

In this study, the MHR was found to be an independent risk factor for the early occurrence of in-hospital cardiovascular adverse events in older adult patients with ACOP myocardial injury. The higher the MHR is, the greater the likelihood of patients developing cardiovascular adverse events. Its predictive value for in-hospital cardiovascular adverse events, as measured by the area under the receiver operating characteristic curve, is 0.797, with a cutoff value of 0.645. The sensitivity was 68.0%, and the specificity was 88.2%.

This study has several limitations. First, as a single-center retrospective cohort study with a limited sample size, it may affect the generalizability of the study results. Second, the study mainly discusses the prediction of in-hospital adverse cardiovascular events by hematological indicators, without obtaining long-term follow-up data, and lacks the ability to predict the MHR for long-term adverse cardiovascular events. In addition, other variables that may affect patient prognosis, such as lifestyle, genetic factors, and the management of complications, were not considered. Therefore, multicenter, large-sample prospective studies with long-term follow-up data are needed to further confirm these findings.

In summary, the new inflammatory indicator MHR was identified as an independent risk factor for predicting in-hospital cardiovascular adverse events in elderly patients with ACOP myocardial injury in this study. These findings provide simple and low-cost methods for assessing the risk of cardiovascular adverse events in older adult ACOP patients, which can help guide clinical decisions and improve patient management and prognosis.

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Conflicts of interest: *The authors report that there are no competing interests to declare.*

Data availability statement: *All data relevant to the study are included in the article or uploaded as an additional file.*

Additional file:

Additional file 1: *STROBE checklist.*

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