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ORIGINAL ARTICLE

Effect of early intervention on short-term prognosis of patients with myocardial injury induced by acute carbon monoxide poisoning

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Abstract

Aim This study aimed to investigate the effect of early intervention on the short-term prognosis of patients with myocardial injury induced by acute carbon monoxide poisoning (ACOP).

Methods and results We performed a retrospective cohort study of 139 patients admitted to the hospital for ACOP-induced acute toxic cardiopathy. Compared with the mild and moderate toxic cardiopathy group, the severe toxic cardiopathy group has significantly increased coma time, acute physiology and chronic health status (APACHE) II score, and the length of hospital stay and significantly reduced proportion of patients with immediate endotracheal intubation and early admission to intensive care unit (ICU) (all $P < 0.05$). The cardiac troponin I (cTnI) levels and corrected QT dispersion (QTcd) duration in three patient groups were significantly higher (all $P < 0.05$) than those in the control group, with the highest in the severely toxic heart disease group. Serum cTnI level and QTcd duration were two independent predictors of myocardial injury in ACOP patients. There was a positive correlation between the APACHE II score and serum cTnI level/QTcd duration at admission. The sensitivities of cTnI and QTcd at admission to diagnose serious cardiovascular events were 78.6% and 85.7%, respectively, and the specificities were both 75%.

Conclusions Acute carbon monoxide poisoning patients with myocardial injury need to be admitted to the hospital as early as possible. For patients with severe hypoxia, an artificial airway should be established as early as possible, and patients should be admitted to the monitoring ward to stabilize their condition at the early stage of poisoning. Meanwhile, changes in QTcd, serum cTnI, and creatine kinase-MB (CK-MB) should be closely observed.

Keywords Acute carbon monoxide poisoning; Myocardial injury; Corrected QT dispersion; Cardiac troponin I; APACHE II score

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Introduction

Acute carbon monoxide poisoning (ACOP) is an important issue in public health worldwide with a high rate of disability and mortality.[1] The mechanism of ACOP is that the affinity of CO and haemoglobin is much higher than that of oxygen and haemoglobin, and the carboxyhaemoglobin is very stable and not easily dissociated, resulting in hypoxia of the whole body and causing damage to multiple organs.[2] As an organ sensitive to hypoxia, the heart is vulnerable during ACOP. As reported, more than one-third of moderate-to-severe ACOP is accompanied by myocardial injury, including angina, myocardial infarction, arrhythmias, and so on.[3] However, effective and objective indicators for judging the condition and prognosis of myocardial injury induced by ACOP are lacking. Therefore, in this study, we conducted a retrospective cohort study for determining the clinical value of various factors in assessing the severity and prognosis of myocardial injury induced by ACOP, hoping to provide the clinical basis for the treatment and prognosis assessment of myocardial injury induced after ACOP.

Materials and methods

Subjects

From January 2018 to October 2020, 139 patients with acute toxic cardiopathy caused by ACOP (69 males and 70 females) from Harrison International Peace Hospital Affiliated to Hebei Medical University were enrolled in this study. Diagnostic criteria of ACOP were based on 'Guidelines for clinical treatment of carbon monoxide poisoning'. The principles of diagnosis are as follows: (i) a history of exposure to higher CO concentrations; (ii) signs and symptoms of acutely occurring central nervous system damage; (iii) combining with the results of timely serum carbon monoxide haemoglobin measurements, on-site sanitary surveys, and airborne carbon monoxide concentration measurements; and (iv) the exclusion of other aetiologies.

Diagnostic criteria of acute toxic cardiopathy were based on 'Diagnostic criteria of occupational acute toxic cardiopathy caused by chemicals' (GBZ74-2009).[4]

Inclusion criteria were as follows: (i) ACOP was diagnosed; and (ii) acute toxic cardiopathy was diagnosed.

Exclusion criteria were as follows: (i) patients poisoned by mixing other poisons; (ii) patients who had chronic basic diseases of heart, brain, liver, kidney, and lung; and (iii) patients with abnormal electrocardiograms (ECGs) due to electrolyte disturbance, temperature, and so forth.

Groups

Take the patient's admission as the starting point of the study, and take the patient's discharge or death as the endpoint of the observation. Based on the 'Diagnostic criteria of occupational acute toxic cardiopathy caused by chemicals' (GBZ74-2009),[4] patients were divided into mild toxic cardiopathy (69 patients), moderate toxic cardiopathy (53 patients), and severe toxic cardiopathy (17 patients).

Mild toxic cardiopathy is diagnosed when one of the following conditions occurs: (i) mild ischaemic changes in ECG; (ii) paroxysmal supraventricular tachycardia or unifocal premature ventricular contraction or type I atrioventricular block; (iii) creatine kinase-MB (CK-MB) levels reached or exceeded the normal reference value by two times but not more than five times, with lactate dehydrogenase (LDH) and glutamic oxalacetic transaminase (AST) increased; and (iv) positive for cardiac troponin I (cTnI).

Moderate toxic cardiopathy is diagnosed when one of the following conditions occurs: (i) significant ischaemic changes in ECG; (ii) paroxysmal ventricular tachycardia or multisource premature ventricular contraction or type II atrioventricular block or atrial fibrillation or atrial flutter; and (iii) CK-MB reached or exceeded five times of the normal reference value, with LDH and AST increased.

Severe toxic cardiopathy is diagnosed when one of the following conditions occurs: (i) myocardial infarction-like changes in ECG; (ii) ventricular fibrillation, ventricular arrest, III degree atrioventricular block, or ventricular tachycardia with tip-twist; (iii) heart failure or cardiogenic shock; and (iv) sudden cardiac death.

The clinical information of each group was exhibited in *Table 1*. Meanwhile, 20 ACOP patients without myocardial injury were enrolled as controls.

Treatment

After admission, all patients received ECG monitoring, oxygen inhalation, prevention of infection, and hyperbaric oxygen therapy. Patients who could not receive hyperbaric oxygen therapy were intermittently given the ventilator to assist with oxygen intake. Patients with poor airway received endotracheal intubation. Mannitol and dexamethasone were supplemented to prevent cerebral oedema. Cytochrome C was supplemented to improve tissue oxygen supply. Coenzyme Q10 was supplemented to provide nutritional support. Amiodarone, magnesium, and metoprolol were given to patients with arrhythmias. Electric shock defibrillation was given to patients with ventricular fibrillation. Diuretics, nitrate, and sodium were given to patients with heart failure. Patients

Table 1 Clinical data of patients enrolled in this study

	Mild group (n = 69)	Moderate group (n = 53)	Severe group (n = 17)	Control group (n = 20)
Age (years)	49.32 ± 1.68	47.59 ± 1.48	48.19 ± 1.76	46.83 ± 1.92
Gender (M/F)	34/35	27/26	8/9	11/9
Body mass index (kg/m^2)	23.54 ± 1.38	22.84 ± 1.67	24.11 ± 1.52	23.46 ± 1.47
Heart rate (b.p.m.)	108.37 ± 4.19	107.39 ± 6.53	110.16 ± 5.38	4
Systolic pressure (mmHg)	147.36 ± 9.17	144.92 ± 7.92	153.29 ± 7.18	6
No treatment period (h)	3.16 ± 0.77	2.89 ± 0.58	3.28 ± 0.61	8
Administration of drugs that affect QT interval (%)	20.29	22.64	23.53	9
HbCO concentration at admission	45.13 ± 8.52	44.13 ± 7.65	43.28 ± 5.68	42.74 ± 5.68

HbCO, carbon monoxide haemoglobin.
Mild group: mild toxic cardiopathy induced by acute carbon monoxide poisoning (ACOP); moderate group: moderate toxic cardiopathy induced by ACOP; severe group: severe toxic cardiopathy induced by ACOP.

who occurred cardiac shock were given aortic balloon counterpulsation [intra-aortic balloon pump (IABP)] or combined with extracorporeal membrane oxygenation (ECMO). Patients with cardiac shock were given cardiopulmonary resuscitation.

Indicators

Time from poisoning to attendance, length of hospital stay, coma time, whether performed endotracheal intubation, whether admission to the intensive care unit (ICU), and acute physiology and chronic health status (APACHE) II score were recorded from all patients. All patients received a 12-lead ECG examination on admission, 2 days, and 3 days after admission, and discharge. QT interval and RR interval were measured as parameters of ventricular repolarization. Corrected QT (QTc) and QTc dispersion (QTcd) intervals were determined with Bazett's formula. Venous blood (10 mL) was collected from all patients at admission, 2 days, and 3 days after admission, and discharge. Serum levels of cTnI and CK-MB were measured by a 7600 automatic biochemical analyser (HITACHI, Japan). Besides, serious cardiovascular events (SCE) during 1 month of poisoning were recorded. SCE is diagnosed when one of the following conditions occurs: (i) acute myocardial infarction; (ii) acute left heart failure; (iii) malignant arrhythmias: ventricular fibrillation, ventricular flutter, polymorphic premature ventricular beats, ventricular tachycardia, third-degree atrioventricular block, and severe sinus arrest; and (iv) sudden cardiac death.

Statistical analysis

Data were analysed using SPSS 23.0. The data are presented as mean ± standard deviation. *T*-test was applied for the comparison between groups. Analysis of variance (ANOVA) for repeated measures was used to compare continuous variables. Independent predictive factors were determined by multivariate analysis. Correlation analysis was analysed by the Spearman and binary logistic regression analysis. $P < 0.05$ was considered statistically significant.

Results

Comparison of hospital course in patients with toxic cardiopathy induced by acute carbon monoxide poisoning

The clinical information of each group was exhibited in *Table 1*. There were no significant differences in gender, age, body mass index, heart rate, systolic pressure, no treatment period, and the administration of drugs that affect QT interval among the groups ($P > 0.05$). As shown in *Table 2*, there was no significant difference between the three groups in the time from poisoning to attendance ($P > 0.05$). Compared with the mild and moderate toxic cardiopathy group, the coma time, APACHE II score, and the length of hospital stay in the severe toxic cardiopathy group were significantly higher ($P < 0.05$). What's more, the proportion of patients with immediate endotracheal intubation and early admission

Table 2 Comparison of hospital course in patients

Variables	Mild group ($n = 69$)	Moderate group ($n = 53$)	Severe group ($n = 17$)	*P*-value
Time from poisoning to attendance (h)	3.27 ± 0.65	3.47 ± 0.82	3.27 ± 0.74	0.28
Performed endotracheal intubation within 3 h of admission (cases)	47 (76%)	33 (58%)	4 (24%)	0.02
Admission to ICU immediately after diagnosis (cases)	44 (64%)	42 (79%)	5 (29%)	0.01
Coma time (h)	9.37 ± 5.25	10.21 ± 4.36	14.49 ± 4.36$^{*,\#}$	0.01
APACHE II score	14.37 ± 2.18	18.27 ± 2.39*	24.63 ± 4.51$^{*,\#}$	0.00
Length of hospital stay (days)	14.69 ± 1.44	15.37 ± 1.48	23.79 ± 1.62$^{*,\#}$	0.00
Intra-aortic balloon pump (cases)	0	0	2 (12%)	0.00
Extracorporeal membrane oxygenation (cases)	0	0	1 (6%)	0.01
Supraventricular tachycardia (cases)	6 (9%)	1 (2%)	0	0.02
Atrial flutter or atrial fibrillation (cases)	0	17 (32%)	8 (47%)	0.01
Ventricular arrhythmias (cases)	0	30 (57%)	8 (47%)	0.00
LVEF at admission (%)	53.86 ± 7.32	51.36 ± 8.14	50.18 ± 8.64	0.57
ALT at admission (U/L)	28.57 ± 6.24	26.81 ± 5.48	29.31 ± 7.75	0.41
Ccr at admission (μmol/L)	63.25 ± 7.83	68.24 ± 6.91	70.13 ± 7.86	0.32

ALT, glutamic-pyruvic transaminase; APACHE II score, acute physiology and chronic health status II score; Ccr, endogenous creatinine clearance rate; ICU, intensive care unit; LVEF, left ventricular ejection fraction.
Mild group: mild toxic cardiopathy induced by acute carbon monoxide poisoning (ACOP); moderate group: moderate toxic cardiopathy induced by ACOP; severe group: severe toxic cardiopathy induced by ACOP.
$^{*}P < 0.05$ vs. mild group.
$^{\#}P < 0.05$ vs. moderate group.

to ICU in the severe toxic cardiopathy group was lower than that in the mild and moderate toxic cardiopathy group ($P < 0.05$). Supraventricular tachycardia mainly occurred in the mild toxic cardiopathy group, while atrial flutter or atrial fibrillation and ventricular arrhythmias mainly occurred in the moderate and severe toxic cardiopathy group.

Mean values of cardiac troponin I and corrected QT dispersion in acute carbon monoxide poisoning patients with different cardiac events

The average cTnI value of ACOP patients with acute myocardial infarction, acute heart failure, ventricular tachycardia, ventricular fibrillation, cardiogenic shock, atrial flutter or atrial fibrillation, ventricular premature beat, and III type atrioventricular block was statistically higher than that of patients in the control group (*Table 3*). The average QTcd value of ACOP patients with acute myocardial infarction, acute heart failure, ventricular tachycardia, ventricular fibrillation, cardiogenic shock, ventricular premature beat, III type atrioventricular block, and sinus bradycardia was statistically longer compared with patients in the control group (*Table 3*).

Comparison of clinical data in patients with toxic cardiopathy induced by acute carbon monoxide poisoning

As shown in *Table 4*, at admission, there was no significant difference in the level of CK-MB of patients in the three groups and controls ($P > 0.05$). Two to 3 days after admis-

Table 3 Comparison of cardiac troponin I and corrected QT dispersion mean values (Days 1–3 after admission) in acute carbon monoxide poisoning patients with different cardiac events

Cardiac events	Cases	Average cTnI value	*P*-value	Average QTcd value	*P*-value
Acute myocardial infarction	8	6.28 ± 1.39	0.000***	57.86 ± 8.37	0.000***
Acute heart failure	27	2.16 ± 0.52	0.014*	48.17 ± 9.26	0.008**
Ventricular tachycardia	10	2.25 ± 0.63	0.023*	58.41 ± 4.62	0.000***
Ventricular fibrillation	3	4.37 ± 1.03	0.001**	75.29 ± 3.46	0.000***
Cardiogenic shock	2	4.28 ± 1.21	0.001**	53.16 ± 8.27	0.009**
Supraventricular tachycardia	7	0.03 ± 0.01	0.142	31.92 ± 4.68	0.087
Atrial flutter or atrial fibrillation	25	0.16 ± 0.04	0.041*	33.08 ± 7.35	0.124
Ventricular premature beat	35	0.18 ± 0.05	0.034*	51.37 ± 6.39	0.003**
III type atrioventricular block	9	0.21 ± 0.05	0.038*	59.26 ± 9.41	0.000***
Sinus bradycardia	19	0.04 ± 0.01	0.256	34.82 ± 3.39	0.001**
Control group	20	0.03 ± 0.01	—	32.23 ± 7.58	—

cTnI, cardiac troponin I; QTcd, corrected QT dispersion.
*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$ vs. controls.

Table 4 Comparison of clinical data in patients

	Mild group ($n = 69$)	Moderate group ($n = 53$)	Severe group ($n = 17$)
At admission			
CK-MB (U/L)	24.38 ± 8.76	23.19 ± 7.14	25.42 ± 5.16
cTnI (μg/L)	0.68 ± 0.41*	2.12 ± 0.78*,#	4.37 ± 1.47*,#,&
QTcd (ms)	48.39 ± 8.59*	61.23 ± 8.46*	68.42 ± 9.41*,#,&
2 days			
CK-MB (U/L)	31.32 ± 6.18*	52.65 ± 7.21*,#	74.26 ± 9.28*,#,&
cTnI (μg/L)	0.89 ± 0.68*	1.53 ± 1.15*,#	3.42 ± 1.39*,#,&
QTcd (ms)	51.54 ± 11.28*	58.54 ± 11.64*,#	63.54 ± 12.86*,#
3 days			
CK-MB (U/L)	34.17 ± 13.92	35.42 ± 13.86	49.32 ± 17.54*,#,&
cTnI (μg/L)	0.42 ± 0.35*	0.78 ± 1.12*,#	1.84 ± 1.36*,#,&
QTcd (ms)	48.75 ± 9.27*	55.74 ± 10.21*,#	59.65 ± 8.26*,#,&
On discharge			
CK-MB (U/L)	23.87 ± 6.04	22.54 ± 4.35	23.17 ± 6.26
cTnI (μg/L)	0.09 ± 0.06	0.27 ± 0.64	0.47 ± 1.32
QTcd (ms)	32.07 ± 6.27	30.77 ± 5.58	34.58 ± 8.26*,#,&

CK-MB, creatine kinase-MB; cTnI, cardiac troponin I; QTcd, corrected QT dispersion.
Control group: CK-MB = 23.15 ± 6.37 (U/L), cTnI = 0.32 ± 0.47 (μg/L), and QTcd = 32.62 ± 8.09 (ms).
*$P < 0.05$ vs. controls.
#$P < 0.05$ vs. mild group.
&$P < 0.05$ vs. moderate group.

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sion, the CK-MB of patients in the severe toxic cardiopathy group was significantly elevated than that of the mild/moderate toxic cardiopathy group and controls ($P < 0.05$). On discharge, the CK-MB of patients in the three groups had decreased to no statistical difference from that of controls ($P > 0.05$).

At admission, the cTnI levels and QTcd duration of patients in the three groups were much higher and longer than that of controls ($P < 0.05$) and the patients in the severe toxic cardiopathy group displayed the highest cTnI level and the longest QTcd duration. Two to 3 days after admission, the cTnI levels and QTcd duration of patients in the three groups were partly decreased and shorten but still significantly higher and longer than that of controls ($P < 0.05$). On discharge, the cTnI levels in the three groups and QTcd duration in mild and moderate groups had decreased to no statistical difference from that of controls ($P > 0.05$), while the QTcd duration of patients in the severe group was still markedly longer than that of the mild/moderate group and controls ($P < 0.05$).

Clinical predictors of myocardial injury in acute carbon monoxide poisoning patients

Binary logistic regression analysis was used to evaluate the correlation between various factors we recorded and the severity of myocardial injury in ACOP patients. cTnI and CK-MB are two recognized markers for cardiac injury. QTcd represents the physiological variability of regional ventricular repolarization. APACHE II score at admission, coma time, and length of hospital stay reflects the severity of the disease. Time from poisoning to attendance reflects the duration of hypoxia, which may also aggravate heart damage. The results in *Table 5* depicted two independent predictors of myocardial injury in ACOP patients: serum cTnI level at admission [odds ratio (OR) = 2.365, $P = 0.005$] and QTcd duration at admission (OR = 1.885, $P = 0.042$).

Correlation analysis of serum cardiac troponin I, creatine kinase-MB, and corrected QT dispersion with acute physiology and chronic health status II score

As shown in *Figure 1*, there was a positive correlation between serum cTnI level at admission and APACHE II score ($r = 0.806$, $P < 0.001$; *Figure 1A*), QTcd duration at admission, and APACHE II score ($r = 0.693$, $P < 0.001$; *Figure 1B*), indicating that the higher serum cTnI and longer QTcd duration at admission, the more serious damage in patients. However, there was no correlation between serum CK-MB level at admission and APACHE II score ($P = 0.269$; *Figure 1C*). Two days after admission, the serum CK-MB level exhibited a positive correlation with the APACHE II score ($r = 0.809$, $P < 0.001$; *Figure 1D*).

Diagnostic performance of cardiac troponin I and corrected QT dispersion at admission in diagnosing serious cardiovascular event in acute carbon monoxide poisoning patients

The receiver operating characteristic (ROC) levels of cTnI and QTcd were drawn (*Figure 2*). The cut-off value of cTnI at admission to diagnose SCE was 1.24 μg/L and the area under the curve (AUC) was 0.762 [95% confidence interval (CI): 0.600–0.924], with a sensitivity of 78.6% and a specificity of 75%. The cut-off value of QTcd at admission to diagnose SCE was 49.5 ms and the AUC was 0.808 (95% CI: 0.627–0.989), with a sensitivity of 85.7% and a specificity of 75%.

The outcome of patients with toxic cardiopathy induced by acute carbon monoxide poisoning

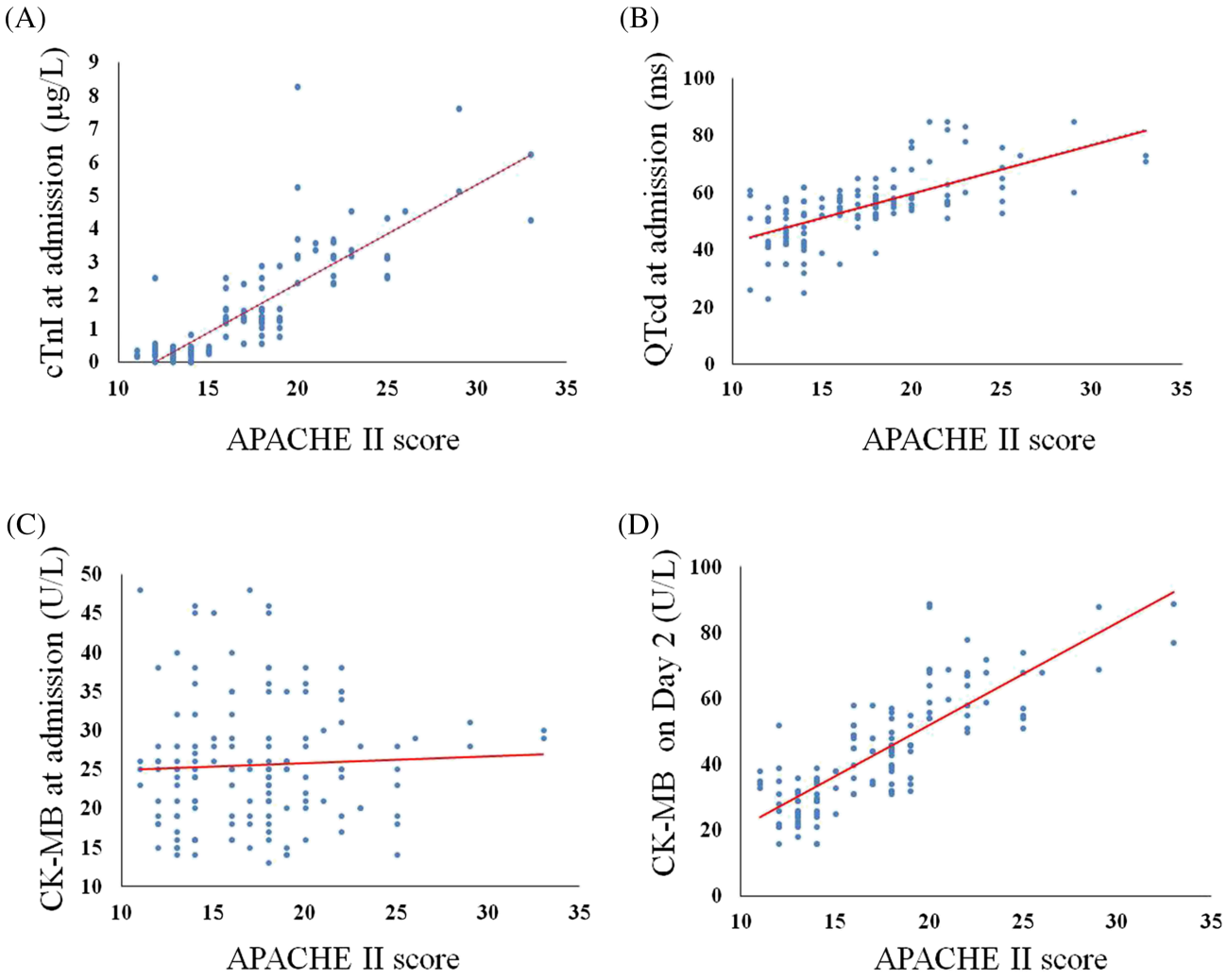
After the treatment, 118 of the 139 patients were discharged from the hospital, 16 cases with delayed encephalopathy were transferred to the rehabilitation department for further

Table 5 Binary logistic regression analysis of various factors and acute carbon monoxide poisoning patients with severe toxic cardiopathy

Variables	*B*	Wald	OR	*P*	95% CI
APACHE II score at admission (score)	0.014	0.016	0.968	0.884	0.769–1.232
cTnI at admission (μg/L)	1.106	8.132	2.365	0.005	1.252–2.801
QTcd at admission (ms)	0.119	3.128	1.885	0.042	0.769–2.013
CK-MB at admission (U/L)	0.028	0.182	1.000	0.636	0.917–1.144
Time from poisoning to attendance (h)	−9.032	0.000	0.000	1.000	0.000
Coma time (h)	−0.055	0.000	0.935	1.000	0.000
Length of hospital stay (days)	6.368	0.000	85.390	0.979	0.000

APACHE II score, acute physiology and chronic health status II score; CI, confidence interval; CK-MB, creatine kinase-MB; cTnI, cardiac troponin I; OR, odds ratio; QTcd, corrected QT dispersion.

Figure 1 Correlation between acute physiology and chronic health status II (APACHE II) score and (A) cardiac troponin I (cTnI) level at admission, (B) corrected QT dispersion (QTcd) duration at admission, (C) creatine kinase-MB (CK-MB) level at admission, and (D) CK-MB level on Day 2 (n = 139).



treatment, and 5 cases died (1 died of acute myocardial infarction, 1 died of cardiogenic shock, 1 died of ventricular fibrillation, and 2 died of severe pneumonia). One of the five deaths was from the moderate toxic cardiopathy group (mortality rate 1.88%) and four were from the severe toxic cardiopathy group (mortality rate 23.52%).

Discussion

In this study, we evaluated the clinical value of various factors in assessing the severity and prognosis of myocardial injury induced by ACOP. The results of our study showed that the serum cTnI level and QTcd duration at admission were positively related to the APACHE II score and could function as potential markers for the severity and short-term prognosis of myocardial injury in ACOP patients.

As shown in the clinical information of each group, the proportion of patients with immediate endotracheal intubation and early admission to ICU in the severe toxic cardiopathy group was lower, which suggested that patients who received timely endotracheal intubation and early admission to ICU had a less severe degree of myocardial damage.

As reported, after ACOP, CO tightly binds to myocardial myoglobin and reduces myocardial oxygen reserve, thus resulting in persistent myocardial damage.[5] Several myocardial injuries, such as atrial fibrillation, premature ventricular beats, and sinus tachycardia, have been observed in patients with ACOP.[6,7] In our study, we enrolled 139 cases of ACOP patients with myocardial injury; among them, ~50% were diagnosed as moderate or severe toxic cardiopathy, indicating

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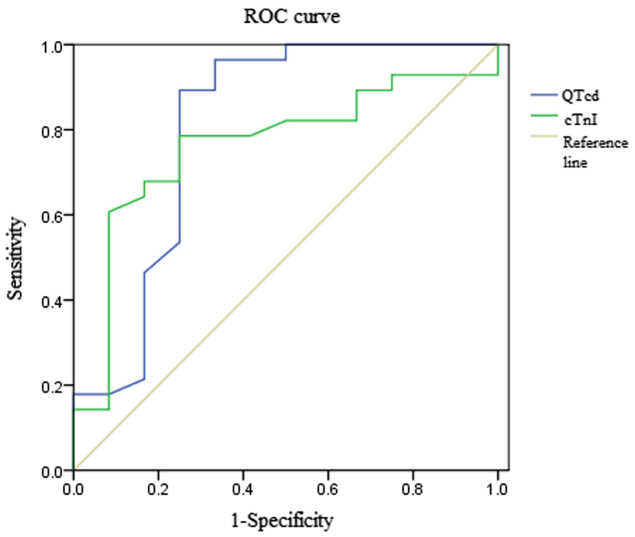
Figure 2 The receiver operating characteristic (ROC) curves of cardiac troponin I (cTnI) and corrected QT dispersion (QTcd) at admission in acute carbon monoxide poisoning patients with serious cardiovascular events.



that the ACOP-induced myocardial injury was widely and severe. Therefore, determining the myocardial damage and evaluating the severity of the damage as early as possible have important guiding significance for the clinical treatment of ACOP.

MtCIV has been reported to be a good marker of ACOP recovery, treatment effectiveness, and the development of advanced neurological syndromes, but it is too sensitive to be a severity marker.[8] cTnI and CK-MB are two recognized markers for cardiac injury.[9,10] Baydin *et al*. found that compared with ACOP patients without cardiac injury, ACOP patients with cardiac injury displayed higher serum levels of cTnI and CK-MB.[11] In line with the previous study, in our study, we found that compared with controls, the serum levels of cTnI in ACOP patients with toxic cardiopathy were elevated at admission, and the ACOP patients with severe toxic cardiopathy showed the highest serum cTnI level. Unlike serum cTnI level, the increase of serum CK-MB levels in ACOP patients with toxic cardiopathy occurred 2 days after admission, indicating that in the early stage of poisoning, serum cTnI is more sensitive than CK-MB. Several studies also supported the higher sensitivity of the cTnI compared with CK-MB in detecting myocardial injury.[12,13] Then, the following study demonstrated that serum level of cTnI at admission was an independent predictor of myocardial injury in ACOP patients. What's more, the serum cTnI level at admission was positively related to the APACHE II score, a reliable marker of physiological impairment.[14] The aforementioned data showed that the serum cTnI level could function as an effective marker for the severity and short-term prognosis of ACOP patients with myocardial injury.

Ventricular repolarization can be evaluated by QT interval, QTc interval, QT dispersion, and QTcd. Among them, QTcd represents the physiological variability of regional ventricular repolarization and its elevation has been proved to be related to ventricular arrhythmia.[15] It has been reported that CO poisoning notably prolonged QTcd in adult patients.[16,17] In agreement with the previous studies, in our study, the duration of QTcd in patients was prolonged after ACOP, and the ACOP patients with severe toxic cardiopathy exhibited the longest QTcd. Of note, binary logistic regression analysis showed that QTcd was an independent predictor of myocardial injury in ACOP patients and there was a positive correlation between the QTcd at admission and APACHE II score, indicating that the prolongation of QTcd could reflect the degree and short-term prognosis of cardiac injury caused by CO poisoning.

Conclusions

Acute carbon monoxide poisoning patients with myocardial injury need to be admitted to the hospital as early as possible. For patients with severe hypoxia, an artificial airway should be established as early as possible, and patients should be admitted to the monitoring ward to stabilize their condition at the early stage of poisoning. Meanwhile, changes in QTcd, serum cTnI, and CK-MB should be closely observed, which is helpful to determine the myocardial damage as soon as possible and evaluate the severity of the damage, and have important guiding significance for the clinical treatment of ACOP.

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Conflict of interest

The authors declare that there is no conflict of interest.

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