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Anticoagulation therapy could improve the restoration of sinus rhythm and spontaneous circulation in hospital patients with CPR

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Abstract

Objective: To analyse the role of anticoagulation therapy in cardiopulmonary resuscitation (CPR) following an in-hospital cardiac arrest.

Methods: This single-centre retrospective cohort study enrolled patients treated with in-hospital CPR that met the inclusion and exclusion criteria. The patients were divided into a without anticoagulation group and an anticoagulation group. The main outcome measures were the restoration of spontaneous respiration, restoration of sinus rhythm (ROSR), restoration of spontaneous circulation (ROSC) and the hospital mortality.

Results: The study analysed 344 patients: 272 in the without anticoagulation group and 72 in the anticoagulation group. Multiple logistic regression analyses demonstrated that anticoagulation therapy improved ROSR (adjusted odds ratio [OR] 2.21, 95% confidence interval [CI] 1.23, 3.96) and ROSC (adjusted OR 1.91, 95% CI 1.08, 3.40), but it did not improve the restoration of spontaneous respiration (adjusted OR 1.64, 95% CI 0.72, 3.76) and hospital survival (adjusted OR 0.90, 95% CI 0.40, 1.99).

Conclusion: Anticoagulation therapy improved ROSR and ROSC, but did not decrease the mortality rate of hospitalized patients undergoing CPR following in-hospital cardiac arrest.

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Anticoagulation, cardiopulmonary resuscitation (CPR), restoration of sinus rhythm (ROSR), restoration of spontaneous circulation (ROSC), hospital mortality

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Introduction

Cardiac arrest is a major international public health problem that accounts for an estimated 15–20% of all deaths.[1] No matter whether it is undertaken in or outside of hospital, the success rate of cardiopulmonary resuscitation (CPR) is very low.[2,3] The main reason is that cessation of blood flow during cardiac arrest results in tissue hypoperfusion, which leads to ischaemia and hypoxia, and finally causes irreversible damage to the heart and brain.[3] At the same time, cessation of blood flow during cardiac arrest can result in microvascular thrombosis, which exacerbates the tissue hypoperfusion and damage to the heart and brain.[3] The irreversible damage to the heart and brain reduces the restoration of sinus rhythm and spontaneous circulation and the survival rates of patients treated with CPR.[4]

An animal experiment found that anticoagulation therapy during CPR significantly reduced the propagation of coagulation, but anticoagulation therapy did not significantly influence the outcome of CPR.[5]Another study found that anticoagulation therapy was not associated with the outcome in patients with out-of-hospital CPR.[6]

There have not been any studies undertaken to study the relationship between anticoagulation therapy and the prognosis of patients treated with CPR in hospital. This study hypothesized that anticoagulation therapy might improve the outcome of patients undergoing CPR. This study investigated the relationship between anticoagulation therapy and outcome of hospital patients treated with CPR.

Patients and methods

Patient population

This single-centre retrospective cohort study enrolled consecutive patients treated with CPR during hospitalization at the Emergency Intensive Care Unit, Second Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi Province, China between January 2011 and December 2016. The exclusion criteria were as follows: (i) age $\leq$ 18 years; (ii) patients that were pregnant; (iii) patients receiving oral anticoagulants; (iv) patients with anticoagulant contraindications (e.g. digestive tract ulcer, gastrointestinal haemorrhage, cerebral haemorrhage and other diseases); (v) patients without key data including anticoagulation therapy, the restoration of sinus rhythm, spontaneous circulation and the final outcome of patients undergoing CPR. The patients were divided into two groups based on whether or not they received anticoagulation therapy before CPR was administered.

Ethical approval was provided by the Ethics Committee of the Second Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi Province, China (no. 20180031). Patients were not required to provide consent to participate due to the

retrospective nature of the study design. All patient data were anonymized.

Anticoagulant therapy

Anticoagulant therapy was defined as parenteral route anticoagulant therapy given within 24 h before CPR after hospital admission. The contraindications for anticoagulation therapy included digestive tract ulcer, gastrointestinal haemorrhage, cerebral haemorrhage and other diseases. The anticoagulation indications included therapeutic anticoagulation and prophylactic anticoagulation. The clinicians comprehensively weighed the contraindications and indications and then decided whether to give anticoagulant therapy. The reasons for administering anticoagulation therapy were recorded for each patient. At the Second Affiliated Hospital of Xi'an Jiaotong University, low-molecular-weight heparin (LMWH) is used most frequently for anticoagulation with fondaparinux and heparin only used rarely (five patients were treated with fondaparinux sodium and three with heparin sodium during the study period), so only patients that received LMWH for anticoagulation were included in the study. The dosage was 4000 IU LMWH sodium (Clexane®; Sanofi, Guildford, UK) every 12 h by subcutaneous injection both for therapeutic and prophylactic anticoagulation.

Outcome measures

The primary outcome measures were the restoration of spontaneous respiration, restoration of sinus rhythm (ROSR), restoration of spontaneous circulation (ROSC) and in-hospital mortality of patients with CPR. The definition of ROSC was as follows: (i) electrocardiogram monitoring showed effective heart rhythm, including sinus, borderline and acceleration of ventricular autonomic rhythm of the heart; (ii) palpable arterial pulsation; (iii) under the condition of spontaneous breathing or mechanical ventilation, with or without drugs to maintain systolic pressure > 60 mmHg.

Statistical analyses

The following were undertaken to control for bias: (i) data collection: a detailed case report form was made before the data were collected, and at the same time, each index was strictly defined to ensure the authenticity and reliability of the data as much as possible; (ii) data management: professional data management software (EpiData Software; The EpiData Association, Odense M, Denmark) was used to manage the data; (iii) data analysis: before data analysis, the data were strictly checked and cleaned; (iv) control of confounding factors: multi-factor analyses were adopted to control for possible confounding factors.

The following were undertaken during the data analysis: (i) data cleaning and interpolation: when patients had missing values or key data, the medical records were checked again and any missing value was supplemented if possible. If there were too many missing values to be supplemented or the key data were still missing, then the patient was excluded. The study only included those patients with a few missing values and those that did not lack the key data; (ii) statistical description: mean $\pm$ SD are used for continuous baseline data in the two groups and categorical data are shown by numerical values and percentages; (iii) univariate analysis: univariate analysis was carried out according to whether anticoagulation therapy was administered. If continuous data met the normal distribution and homogeneity of variance, then the data were compared using *t*-test. If the continuous data did not meet the normal distribution or homogeneity of variance, Mann–Whitney *U*-test was used. χ^2-test was used to compare categorical data; (iv)

multifactor regression analysis was used to further detect the relationship between anticoagulation therapy and the prognosis of hospital patients with CPR. In the multifactor analysis, variables that demonstrated a P-value < 0.05 in the univariate analysis and that were clearly related to the outcome of patients with CPR were adjusted. All statistical analyses were performed using EmpowerStats version number 2018-05-05 (Copyright 2009; X&Y Solutions, Boston, USA). A $P < 0.05$ was considered statistically significant.

Results

This single-centre retrospective cohort study identified 414 patients that underwent in-hospital CPR and of these 344 patients met the inclusion criteria. Figure 1 presents the flow of patients through the study. The patients were divided into two groups based on their treatment prior to CPR: without anticoagulation group ($n = 272$) and with anticoagulation group ($n = 72$). The reasons for using therapeutic anticoagulation ($n = 45$) included atrial fibrillation ($n = 10$), pulmonary embolism ($n = 2$), acute coronary syndrome ($n = 30$), deep venous thrombosis (DVT; $n = 3$); and prophylaxis anticoagulation ($n = 27$) was used for patients with a high risk of DVT, such as trauma ($n = 3$), fracture ($n = 4$), sepsis ($n = 5$), malignant tumour ($n = 3$), chronic heart failure ($n = 5$), chronic obstructive pulmonary disease ($n = 2$) and other risk factors ($n = 5$).

There were no significant differences between the two groups in terms of age, sex, diabetes mellitus, hypertension, atrial fibrillation, cerebral infarction, beta blocker use, smoking and drinking (Table 1). There were no significant differences between the two groups in the amount of epinephrine used, the amount of atropine used, glucocorticoid use, naloxone use, vasopressin use, dobutamine use, number of defibrillations, norepinephrine use, $NaCHO_3$ use, dopamine use, amiodarone use, lidocaine use and tracheal intubation use. A significantly higher proportion of patients in the anticoagulation group had coronary heart disease ($P = 0.001$), received antiplatelet therapy ($P < 0.001$), received isoprenaline

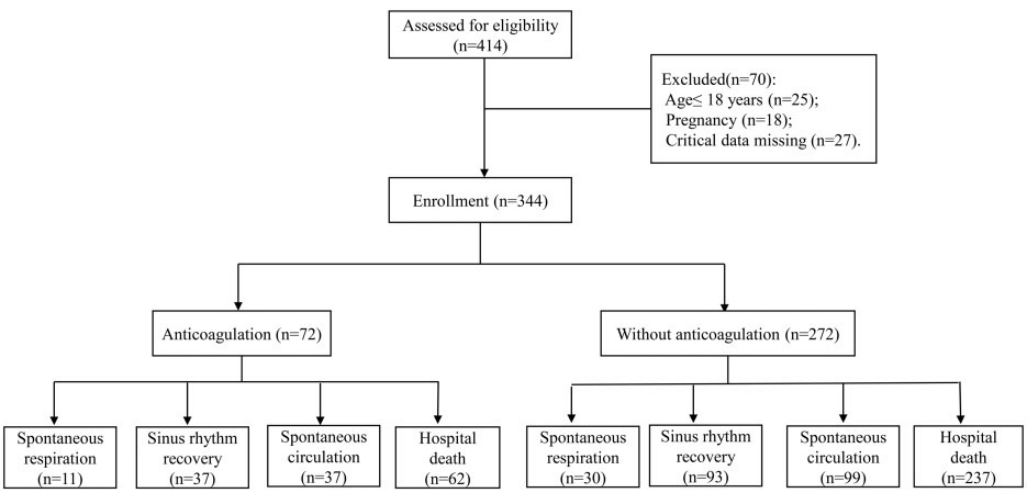

Figure 1. Patient flowchart showing the flow of patients through a single-centre retrospective cohort study that investigated the relationship between anticoagulation therapy and outcome of hospital patients treated with cardiopulmonary resuscitation (CPR).

($P = 0.008$), achieved ROSR ($P = 0.007$) and achieved ROSC ($P = 0.02$) compared with the without anticoagulation group.

The results of the univariate analyses demonstrated that anticoagulant therapy did not improve restoration of spontaneous respiration (odds ratio [OR] 1.52, 95% confidence interval [CI] 0.72, 3.22) and did not decrease in-hospital mortality (OR 0.92, 95% CI 0.43, 1.95) (Tables 2 and 3). Anticoagulant therapy did improve ROSR (OR 2.03, 95% CI 1.20, 3.44, $P = 0.008$) and ROSC (OR 1.85, 95% CI 1.09, 3.12, $P = 0.02$).

The multiple logistic regression analyses demonstrated that anticoagulation therapy improved ROSR (adjusted OR 2.21, 95% CI 1.23, 3.96, $P = 0.04$) and ROSC

Table 1. Baseline demographic and clinical characteristics, intervention and outcome for patients enrolled in a single-centre retrospective cohort study that investigated the relationship between anticoagulation therapy and outcome of hospital patients treated with cardiopulmonary resuscitation.

Characteristic	Without anticoagulation group $n = 272$	With anticoagulation group $n = 72$	Statistical significance[a]
Age, years	67.93 ± 17.91	66.49 ± 16.68	NS
Sex, male/female	170/102	41/31	NS
Diabetes mellitus	62 (22.79%)	20 (27.78%)	NS
Hypertension	117 (43.01%)	34 (47.22%)	NS
Coronary heart disease	94 (34.56%)	40 (55.56%)	$P = 0.001$
Atrial fibrillation	34 (12.50%)	12 (16.67%)	NS
Cerebral infarction	54 (19.85%)	9 (12.50%)	NS
Beta blocker use	3 (1.10%)	3 (4.17%)	NS
Smoker	75 (27.57%)	18 (25.00%)	NS
Alcohol drinker	30 (11.03%)	8 (11.11%)	NS
Antiplatelet therapy	52 (19.12%)	46 (63.89%)	$P < 0.001$
Amount of epinephrine, mg	6.17 ± 4.71	6.28 ± 5.29	NS
Amount of atropine, mg	2.74 ± 2.15	2.88 ± 2.04	NS
Glucocorticoid use	57 (20.96%)	15 (20.83%)	NS
Naloxone use	49 (18.01%)	11 (15.28%)	NS
Vasopressin use	1 (0.37%)	1 (1.39%)	NS
Dobutamine use	10 (3.68%)	7 (9.72%)	NS
Number of defibrillations	0.47 ± 1.17	0.53 ± 1.10	NS
Isoprenaline use	45 (16.54%)	22 (30.56%)	$P = 0.008$
Norepinephrine use	71 (26.10%)	12 (16.67%)	NS
$NaHCO_3$ use	166 (61.03%)	48 (66.67%)	NS
Dopamine use	118 (43.38%)	38 (52.78%)	NS
Amiodarone use	32 (11.76%)	13 (18.06%)	NS
Lidocaine use	29 (10.66%)	11 (15.28%)	NS
Tracheal intubation use	218 (80.15%)	59 (81.94%)	NS
Restoration of spontaneous respiration	30 (11.03%)	11 (15.28%)	NS
Restoration of sinus rhythm	93 (34.19%)	37 (51.39%)	$P = 0.007$
Restoration of spontaneous circulation	99 (36.40%)	37 (51.39%)	$P = 0.02$
Hospital death	237 (87.13%)	62 (86.11%)	NS

Data presented as mean $\pm$ SD or *n* of patients (%).

[a]Between-group comparison: continuous data were compared using *t*-test or Mann–Whitney *U*-test; categorical data were compared using χ^2-test; NS, no significant between-group difference ($P \geq 0.05$).

Table 2. Univariate analyses for restoration of spontaneous respiration and sinus rhythm in patients enrolled in a single-centre retrospective cohort study that investigated the relationship between anticoagulation therapy and outcome of hospital patients treated with cardiopulmonary resuscitation.

Characteristic	Restoration of spontaneous respiration OR (95% CI), *P*-value	Restoration of sinus rhythm OR (95% CI), *P*-value
Age	0.98 (0.97, 1.00), NS	1.00 (0.98, 1.01), NS
Sex, male	1	1
Sex, female	0.55 (0.26, 1.13), NS	1.04 (0.66, 1.63), NS
Diabetes mellitus	0.79 (0.35, 1.79), NS	1.22 (0.74, 2.03), NS
Hypertension	1.00 (0.52, 1.93), NS	0.86 (0.55, 1.33), NS
Coronary heart disease	0.69 (0.34, 1.39), NS	1.13 (0.72, 1.76), NS
Atrial fibrillation	0.68 (0.23, 2.01), NS	1.17 (0.62, 2.20), NS
Cerebral infarction	0.96 (0.40, 2.28), NS	1.40 (0.81, 2.44), NS
Beta blocker use	1.44 (0.16, 12.69), NS	0.82 (0.15, 4.54), NS
Smoking	0.83 (0.39, 1.78), NS	0.82 (0.50, 1.35), NS
Alcohol drinking	0.86 (0.70, 1.04), NS	0.94 (0.83, 1.07), NS
Antiplatelet therapy use	1.39 (0.69, 2.78), NS	1.52 (0.94, 2.44), NS
Anticoagulant therapy use	1.52 (0.72, 3.22), NS	2.03 (1.20, 3.44), $P = 0.008$
Amount of epinephrine	0.88 (0.79, 0.97), $P = 0.01$	0.90 (0.85, 0.95), $P < 0.001$
Amount of atropine	0.80 (0.66, 0.97), $P = 0.02$	0.88 (0.79, 0.99), $P = 0.03$
Glucocorticoid use	0.89 (0.39, 2.02), NS	1.14 (0.67, 1.94), NS
Naloxone use	0.78 (0.31, 1.95), NS	0.79 (0.44, 1.42), NS
Vasopressin use	7.32 (0.45, 119.43), NS	1.65 (0.10, 26.63), NS
Dobutamine use	1.03 (0.22, 4.68), NS	2.46 (0.91, 6.64), NS
Dopamine use	0.76 (0.39, 1.49), NS	1.05 (0.68, 1.63), NS
Number of defibrillations	0.91 (0.66, 1.25), NS	0.81 (0.65, 1.01), NS
Isoprenaline use	0.99 (0.43, 2.25), NS	1.14 (0.66, 1.97), NS
Norepinephrine use	0.90 (0.41, 1.98), NS	0.91 (0.55, 1.52), NS
$NaHCO_3$ use	0.95 (0.48, 1.85), NS	0.91 (0.58, 1.42), NS
Amiodarone use	0.71 (0.24, 2.09), NS	1.11 (0.59, 2.11), NS
Lidocaine use	0.80 (0.27, 2.38), NS	1.98 (1.02, 3.84), $P = 0.04$
Tracheal intubation use	0.63 (0.30, 1.33), NS	1.03 (0.59, 1.78), NS

OR, odds ratio; CI, confidence interval; NS, no significant association ($P \geq 0.05$).

(adjusted OR 1.91, 95% CI 1.08, 3.40, $P = 0.03$) (Table 4). Anticoagulation therapy did not improve the restoration of spontaneous respiration (adjusted OR 1.64, 95% CI 0.72, 3.76) and in-hospital survival (adjusted OR 0.90, 95% CI 0.40, 1.99).

Discussion

This current study demonstrated that anticoagulation therapy could improve the restoration of sinus rhythm and the restoration of spontaneous circulation, but it did not improve the restoration of spontaneous respiration and in-hospital survival.

This current study excluded patients that had been administered oral anticoagulants during the study period because these usually take effect slowly, so there was no guarantee that they would have taken effect 24 hours before a cardiac arrest occurred. In addition, patients treated with non-LMWH anticoagulants were excluded

Table 3. Univariate analyses for restoration of spontaneous circulation and hospital death in patients enrolled in a single-centre retrospective cohort study that investigated the relationship between anticoagulation therapy and outcome of hospital patients treated with cardiopulmonary resuscitation.

Characteristic	Restoration of spontaneous circulation OR (95% CI), *P*-value	Hospital mortality OR (95% CI), *P*-value
Age	1.00 (0.99, 1.01), NS	1.01 (0.99, 1.02), NS
Sex, male	1	1
Sex, female	0.97 (0.62, 1.51), NS	0.84 (0.45, 1.59), NS
Diabetes mellitus	1.04 (0.63, 1.72), NS	1.52 (0.68, 3.41), NS
Hypertension	0.88 (0.57, 1.35), NS	1.49 (0.78, 2.86), NS
Coronary heart disease	1.17 (0.75, 1.82), NS	0.46 (0.21, 1.02), NS
Atrial fibrillation	0.98 (0.52, 1.85), NS	0.99 (0.39, 2.48), NS
Cerebral infarction	1.28 (0.74, 2.23), NS	1.04 (0.46, 2.36), NS
Beta blocker use	0.76 (0.14, 4.21), NS	–
Smoking	0.65 (0.39, 1.08), NS	1.02 (0.50, 2.07), NS
Alcohol drinking	1.00 (0.50, 1.99), NS	0.99 (0.37, 2.69), NS
Antiplatelet therapy use	1.53 (0.95, 2.46), NS	0.68 (0.35, 1.33), NS
Anticoagulant therapy use	1.85 (1.09, 3.12), $P = 0.02$	0.92 (0.43, 1.95). NS
Amount of epinephrine	0.91 (0.86, 0.96), $P < 0.001$	1.06 (0.98, 1.14), NS
Amount of atropine	0.89 (0.80, 0.99), $P = 0.03$	1.35 (1.11, 1.64), $P = 0.003$
Glucocorticoid use	0.97 (0.57, 1.65), NS	0.61 (0.30, 1.22), NS
Naloxone use	0.72 (0.40, 1.30), NS	1.80 (0.68, 4.78), NS
Vasopressin use	1.53 (0.10, 24.72), NS	–
Dobutamine use	2.28 (0.85, 6.14), NS	0.25 (0.09, 0.71), $P < 0.001$
Dopamine use	1.12 (0.73, 1.73), NS	1.16 (0.61, 2.18), NS
Number of defibrillations	0.89 (0.73, 1.09), NS	1.17 (0.84, 1.64), NS
Isoprenaline use	1.04 (0.60, 1.79), NS	0.82 (0.39, 1.76), NS
Norepinephrine use	1.01 (0.61, 1.68), NS	0.98 (0.47, 2.03), NS
$NaHCO_3$ use	0.92 (0.59, 1.44), NS	1.00 (0.52, 1.91), NS
Amiodarone use	1.26 (0.67, 2.38), NS	0.46 (0.21, 1.02), NS
Lidocaine use	1.62 (0.84, 3.14), NS	0.55 (0.24, 1.29), NS
Tracheal intubation use	1.04 (0.60, 1.80), NS	1.84 (0.91, 3.74), NS

OR, odds ratio; CI, confidence interval; NS, no significant association ($P \geq 0.05$).

from this study because very few patients were administered these anticoagulants during the study period. By excluding patients treated with oral anticoagulants and non-LMWH, this study reduced the risk of potential confounding factors. Furthermore, when rescuing patients experiencing cardiac arrest the administration of oral medication is particularly challenging, so parenteral route anticoagulant therapy was much easier to administer.

Cessation of blood flow during cardiac arrest can result in ischaemia, hypoxia and acidosis.[4] The subsequent cardiopulmonary resuscitation can lead to ischaemia–reperfusion injury of tissues,[7] which can lead to vascular endothelial cell injury that initiates coagulation reactions and systemic inflammatory reactions.[8] These in turn can result in blood coagulation disorders, microvascular thrombosis, disseminated intravascular coagulation (DIC) and multiple organ failure.[9] These may be the main reasons for the poor results of CPR. Therefore, anticoagulation therapy may be helpful to improve the outcome after CPR.

Table 4. Multiple logistic regression analyses in patients enrolled in a single-centre retrospective cohort study that investigated the relationship between anticoagulation therapy and outcome of hospital patients treated with cardiopulmonary resuscitation.

Exposure	Not-adjusted OR (95% CI), *P*-value	Adjusted OR[a] (95% CI), *P*-value
Restoration of spontaneous respiration		
Without anticoagulant	1	1
With anticoagulant	1.52 (0.72, 3.22), NS	1.64 (0.72, 3.76), NS
Restoration of sinus rhythm		
Without anticoagulant	1	1
With anticoagulant	2.03 (1.20, 3.44), $P = 0.008$	2.21 (1.23, 3.96), $P = 0.04$
Restoration of spontaneous circulation		
Without anticoagulant	1	1
With anticoagulant	1.85 (1.09, 3.12), $P = 0.02$	1.91 (1.08, 3.40), $P = 0.03$
Hospital death		
Without anticoagulant	1	1
With anticoagulant	0.92 (0.43, 1.95), NS	0.90 (0.40, 1.99), NS

[a]Adjusted variables: age, sex, number of defibrillations electric defibrillation time, amount of atropine used, amount of epinephrine used, coronary heart disease, antiplatelet therapy and giving up therapy.
OR, odds ratio; CI, confidence interval; NS, no significant association ($P \geq 0.05$).

There are several related studies that have shown that coagulation dysfunction is very common during or after CPR and that microvascular thrombosis was the most common complication.[10,11] Other studies have shown that anticoagulation after CPR improved the final outcome of patients treated with CPR. For example, a study that included 317 post-CPR patients found that patients with DIC had a worse neurological outcome (OR 1.8, 95% CI 1.323, 2.451) and a higher 6-month mortality rate (OR 1.7, 95% CI 1.307, 2.405).[12] A retrospective study that included 315 patients found that DIC scores and fibrin/fibrinogen degradation product levels were associated with neurological outcomes.[13] D-dimer concentration was found to be an independent predictor of all-cause mortality in patients after CPR.[14] A prospective study showed that the D-dimer concentration in patients after CPR was significantly higher than that in patients without CPR,[15] indicating that thromboembolism occurred during CPR. Meanwhile, the study also found that anticoagulation after CPR could significantly improve the prognosis of patients with CPR.[15] These findings suggest that thrombosis after CPR is a very common complication and that anticoagulation therapy after CPR could improve the prognosis of patients with CPR.[12–15]

There has only been one animal study and one retrospective human study on the use of anticoagulation therapy before CPR. A randomized, blinded animal study investigated the role of anticoagulation therapy in CPR in female domestic pigs.[5] However, this animal study did not explore the effects of anticoagulant on the restoration of sinus rhythm and spontaneous circulation and the prognosis of patients with CPR.[5] As the study only included a small number of animals, the reliability and clinical applicability were very low.[5] A study in patients treated with out-of-hospital CPR found that antiplatelet therapy reduced post-arrest illness severity (adjusted OR 0.50, 95% CI 0.33, 0.77) and was associated with higher survival rates (adjusted OR

1.74, 95% CI 1.08, 2.80) and greater odds of a favourable functional outcome (adjusted OR 2.11 95% CI 1.17, 3.79), but anticoagulation therapy was not associated with illness severity, survival to discharge or favourable outcomes.[6] The possible reasons for these findings being different to those of the current study are as follows: (i) the restoration of sinus rhythm and spontaneous circulation are two of the most important signs of the success of CPR, but this previous study didn't include these parameters;[6] (ii) the two clinical studies had different study populations, with the cases included in the previous study being patients with out-of-hospital cardiac arrest,[6] but the patients in the current study experienced in-hospital cardiac arrest. The survival to discharge and favourable outcomes were consistent with this current study, which suggest that the conclusion is reliable. At the same time, the patients in the current study had in-hospital cardiac arrests, so this current research has some clinical value in the treatment of patients with in-hospital cardiac arrest.

This current study found that anticoagulation therapy prior to CPR improved the restoration of sinus rhythm and the restoration of spontaneous circulation. Additional multi-factor analysis that adjusted for possible confounding variables reached the same conclusion, which suggests that the conclusions from this current study are reliable. The current study used an adjusted model to control for the potential confounding factors, making the conclusions more reliable.[16]

In terms of the generalizability of this current study: (i) this study was one of a few studies to explore the effect of anticoagulant therapy in hospitalized patients with CPR; (ii) during the process of this research study, both the data collection and the statistical processes were designed to control the risk of bias; (iii) this study found that anticoagulation improved the restoration of sinus rhythm and spontaneous circulation in hospitalized patients with CPR so it may provide a potential treatment method for CPR and may improve the success rate of CPR; (iv) in our opinion, for patients with a high risk of cardiac arrest, anticoagulation therapy should be administered after excluding relevant contraindications as this might provide potential benefits; (v) the results of this current study have provided new ideas for further research. For example, this current study examined the use of anticoagulant therapy prior to cardiac arrest and CPR, but whether anticoagulant therapy used during CPR would improve the prognosis of patients needs further investigation.

This study had several limitations. First, it was a retrospective study with a relatively small study population, so its conclusions need to be further verified in a prospective study with a larger sample size. Secondly, the patients included in this study were all in-hospital cases, which would limit their clinical applicability. Thirdly, the arrest rhythm was not be obtained at the beginning of the cardiac arrest. Finally, there were some missing values in the research data.

In conclusion, anticoagulation therapy improved the restoration of sinus rhythm and spontaneous circulation, but did not improve the restoration of spontaneous respiration and hospital survival, when administered before patients experienced an in-hospital cardiac arrest and CPR.

Declaration of conflicting interest

The authors declare that there are no conflicts of interest.

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