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Efficacy of hemadsorption combining with cardiopulmonary bypass used in patients with acute type A aortic dissection

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Abstract

Background It is hypothesized that combining HA380 with cardiopulmonary bypass (CPB) in acute type A aortic dissection (ATAAD) surgery could reduce the inflammatory response induced by CPB and subsequently improve prognosis.? Therefore, we aimed to assess the short-term effectiveness of combining HA380 with CPB in treating ATAAD.

Methods This study exclusively included individuals diagnosed with ATAAD at our institution from January 2021 to April 2023. After propensity score matching (PSM), patients were allocated into two groups: the hemadsorption (HA) group ($n=45$) and the control group ($n=45$). The outcome measures included commonly used clinical inflammatory markers, coagulation function, liver and kidney function, ventilator time, and time of ICU stay duration.

Results Patients in the HA group exhibited elevated postoperative levels of procalcitonin and systemic coagulation-inflammation index (SCI), along with lower white blood cell counts and blood urea nitrogen levels compared to the control group. The HA group had a significantly higher 60-day postoperative survival rate compared to the control group. While the HA group experienced reduced in-hospital mortality and stroke incidence, only the reduction in stroke incidence showed a significant association with the intervention after adjusting for confounders.

Conclusion The application of HA380 does not significantly reduce all postoperative inflammatory indicators in ATAAD surgery. The reduction in inflammatory response was also not obvious. However, it was associated with a significantly lower stroke incidence and improved 60-day survival, suggesting potential clinical benefits in specific outcomes. The specific mechanisms underlying the lower rates of stroke and mortality require further investigation.

Keywords Acute type A aortic dissection, Hemadsorption, Cardiopulmonary bypass, Inflammatory response

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Introduction

Acute type A aortic dissection (ATAAD) triggers a severe systemic inflammatory response during the perioperative period due to circulatory disorders, ischemia-reperfusion injury, cardiopulmonary bypass (CPB), and surgical trauma. This response is characterized by elevated levels of inflammatory cytokines (e.g., IL-6, TNF-α, CRP, MMP-9) and endotoxins [1], which can lead to complications such as arrhythmias, hypotension, stroke, renal failure, and even death [2, 3]. Hemoperfusion has been explored as a method to reduce postoperative inflammation by adsorbing inflammatory factors during CPB, though its efficacy remains inconsistent [4–8]. Patients with significant preoperative inflammation, such as those with endocarditis or sepsis, or those undergoing complex surgeries, may benefit more from hemoperfusion [9]. A meta-analysis [10] further supports its role in reducing mortality in non-elective surgeries, particularly infective endocarditis. Given that ATAAD generates more inflammatory factors due to multi-organ ischemia-reperfusion injury, investigating hemadsorption in ATAAD management is highly relevant.

The systemic immune inflammation index (SII), calculated by multiplying the number of neutrophils by the number of platelets and dividing by the number of lymphocytes; the systemic coagulation-inflammation index (SCI), defined as the number of platelets multiplied by the number of fibrinogens and dividing by the number of white blood cells; and the systemic inflammatory response index (SIRI), represented by the number of monocytes multiplied by the number of neutrophils and divided by the number of lymphocytes, are three inflammation markers that demonstrate strong predictive capabilities regarding the short-term prognosis of diagnosed with ATAAD [11–15]. We collected these relevant inflammatory markers before and after surgery to evaluate the effects of hemadsorption on inflammation.

The HA380 hemoperfusion device, featuring a biocompatible resin-coated polystyrene adsorbent, effectively removes circulating molecules (10–60 kDa) [16]. It has been shown to reduce inflammatory cytokines (e.g., IL-6, IL-10, TNF-α) and improve outcomes in conditions such as sepsis, COVID-19, and post-CPB hemodynamics [17–20]. However, its use in ATAAD surgery has not been studied. This study aims to evaluate the short-term efficacy of HA380 hemadsorption during CPB for ATAAD.

Methods

Study subjects

The participants in the study included individuals diagnosed with ATAAD who underwent open-heart surgery from January 2021 to April 2023. The HA380 hemadsorption device was introduced in April 2022 and has been used in ATAAD surgeries ever since. The inclusion criteria for patients were as follows: (1) ATAAD as the primary diagnosis as defined by the 2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease [21]; (2) aortic enhanced CT showing involvement of the ascending aorta, aortic arch, and part of the descending aorta in the dissection; and (3) the patient undergoing surgical procedures to repair the ascending aorta and aortic arch, as well as the implantion of a descending aorta elephant trunk stent. The exclusion criteria included: (1) patient aged below 18 years or above 80 years; (2) patient undergoing concurrent valve replacement or coronary artery bypass surgery; (3) a previous history of abnormal liver, kidney, or coagulation function; (4) pre-existing tissue or organ infectious diseases; and (5) pregnancy. The patients were classified into two distinct groups: the HA group and the control group, based on whether the HA380 device was used during CPB. To address confounding factors arising from differences in baseline characteristics, propensity score matching was employed. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki and received approval from our institution's Ethics Committee.

CPB combined with hemadsorption using a HA380 cartridge

Both groups of patients were anesthetized using a combination of midazolam, etomidate, rocuronium, and sufentanil, along with propofol, cisatracurium, and remifentanil; anesthesia was maintained through the inhalation of sevoflurane. The CPB procedure utilized a heart-lung machine (Stockert S3) sourced from Stockert in Munich, Germany, and a Medtronic Affinity NT oxygenator manufactured by Medtronic Inc. in Minneapolis, MN, USA. The CPB circuit was primed with a solution consisting of 500 mL of lactated Ringer's, 1000 mL of Polygelatin peptide, and 50 mg of heparin. Heparin was administered at a dose of 4 mg/kg to ensure systemic anticoagulation. The rate of blood perfusion was sustained within a range of 60 to 80 mL/kg/min. During the operation, antegrade selective cerebral perfusion (ASCP) was used during circulatory arrest to ensure adequate cerebral oxygenation. The target nasopharyngeal temperature during circulatory arrest was maintained between 25 °C and 28 °C, depending on the patient's condition and surgical complexity.systolic blood pressure was maintained between 50 and 80 mm Hg, hematocrit levels were kept between 22% and 30%, and the active clotting time exceeding 480 s. When discontinuing CPB, protamine was utilized to counteract the effects of heparin. Hemiarch replacement was performed in the majority of cases, while total arch replacement was reserved for patients with extensive dissection involving the distal arch. In the HA group, a hemoperfusion cartridge (HA380) from

Jafron Biomedical Co. (Zhuhai, China) was employed into the CPB circuit. The blood flow rate through the HA380 cartridge was regulated to remain between 200 and 300 mL/min, and the perfusion duration matched the duration of CPB. In contrast, the control group did not employ the HA380 hemoperfusion cartridge.

Outcome measures

The main outcome measures included the following: (1) inflammatory markers (white blood cell count, platelet count, monocyte count, C-reactive protein, procalcitonin, neutrophil-to-lymphocyte ratio, SII, SCI, and SIRI); (2) coagulation parameters (prothrombin time, activated partial thromboplastin time, and D dimer); and (3) biochemical indexes [creatinine, blood urea nitrogen (BUN), alanine aminotransferase, and aspartate aminotransferase]. The secondary outcome measures included ventilator time, ICU stay duration, length of hospital stay, total hospitalization costs, postoperative renal dialysis, stroke incidence, and in-hospital mortality. Blood samples were collected at four time points: T_1 (return to ICU), T_2 (1 day post-surgery), T_3 (2 days post-surgery), and T_4 (3 days post-surgery).

Statistical analysis

In order to investigate the impact of HA380 on outcome measures, we used propensity score matching (PSM) to ensure a balanced distribution of baseline characteristics between the two groups. The matching tolerance level was set 0.02. The prediction probability was calculated based on several factors, including age, gender, body mass index (BMI), smoking habits, preoperative consciousness levels, limb ischemia, hypertension, and diabetes. we used two-way repeated measures ANOVA to compare the changes in these parameters across different time points. For datasets following a normal distribution, continuous variables were expressed as mean ± standard deviation, whereas median and interquartile range were employed for data with skewed distributions. Categorical variables were summarized as counts and percentages (%). To compare the results between the control and HA groups, an independent sample T-test was used for normally distributed date, and the Mann–Whitney U test was applied for no-normally distributed date. For binary categorical data, the Chi-square test was utilized. Generalized estimating equations were used to analyze differences in repeated measures across groups, and binary logistic regression was performed to investigate the association of interventions and confounders with stroke and in-hospital mortality. A two-tailed p-value < 0.05 was considered statistically significant. The difference in survival rates between the two groups was evaluated through Kaplan-Meier survival analysis. All statistical analyses were conducted using SPSS software version 25 (SPSS Inc., Chicago, IL, USA).

Results

Baseline characteristics of the patient and the procedure

Following the screening, 151 patients were identified. After propensity score matching (PSM), 45 individuals were allocated to the HA group, and another 45 were designated to the control group. No significant variations in baseline characteristics, such as age, sex, BMI, smoking history, preoperative unconsciousness, limb ischemia, hypertension, and diabetes, were observed between the two groups. (Table 1). All patients underwent replacement of the ascending aorta and aortic arch, along with implantation of a descending aorta elephant trunk stent. The duration of CPB was reduced in the HA group compare to the control group [181 (24) vs. 190 (52), $P < 0.05$], and the circulatory arrest temperature was higher in the HA group than in the control group [30.10 (1.50) vs. 29.20 (1.60), $P < 0.05$]. (Table 1).

Table 1 Differences in the baseline characteristics and surgical procedures between the two groups

	Control ($n = 45$)	HA ($n = 45$)	P-value
Female	14 (31.11%)	10 (22.22%)	0.475
Age, years	56.38 ± 10.54	54.67 ± 13.13	0.497
BMI	27.69 ± 3.08	26.95 ± 3.16	0.263
Smoker	18 (40%)	17 (37.78%)	1.000
Unconsciousness	1 (2.22%)	1 (2.22%)	1.000
Hypertension	41 (91.11%)	42 (93.33%)	1.000
Diabetes	1 (2.22%)	1 (2.22%)	1.000
White blood cell, 10^9/L	12.21 ± 4.67	12.11 ± 3.72	0.906
Blood urea nitrogen, mmol/L	6.85 ± 1.90	9.23 ± 13.76	0.263
CPB time, min	190 (52)	181 (24)	0.005*
Circulatory arrest time, min	4 (3)	4 (2)	0.713
Circulatory arrest temperature	29.20 (1.60)	30.10 (1.50)	< 0.001*

*: $P < 0.05$

Primary outcomes

Taking CPB time and circulatory arrest temperature as confounding factors, we evaluated the primary outcome measures for patients in both the HA group and the control group (Table 2). Postoperative levels of WBC and BUN were lower in the HA group compared to the control group. Nevertheless, the levels of procalcitonin (PCT) and SCI were higher in the HA group than in the control group ($P < 0.05$). No statistically significant differences were observed in the coagulation parameters or biochemical markers between the two groups. The trends over time for WBC, BUN, PCT, and SCI are illustrated in Fig. 1.

Table 2 Comparison of the main outcome in the HA group and control group at T_1, T_2, T_3, and T_4

	Control				HA				B(95%CI)	*P*-value
	T_1	T_2	T_3	T_4	T_1	T_2	T_3	T_4		
WBC (10^9/L)	10.58 ± 2.91	13.68 ± 4.40	14.27 ± 3.90	13.21 ± 4.23	8.74 ± 2.92	12.76 ± 4.00	13.36 ± 3.47	11.95 ± 2.88	1.22 (0.06 to 2.39)	0.040*
PLT (10^9/L)	114.00 (58.50)	92.00 (55.00)	73.00 (47.00)	77.00 (63.25)	97.00 (57.00)	90.00 (55.00)	91.00 (49.50)	95.50 (65.25)	-3.46 (19.64 to 12.71)	0.675
Monocytes (10^9/L)	0.72 (0.40)	0.63 (0.56)	0.64 (0.50)	0.67 (0.40)	0.60 (0.43)	0.60 (0.41)	0.70 (0.30)	0.60 (0.20)	0.04 (-0.06 to 0.15)	0.426
PCT (ng/mL)	4.37 (6.11)	26.00 (38.42)	16.68 (33.94)	9.11 (27.28)	6.69 (10.31)	40.72 (89.60)	25.89 (36.20)	12.19 (22.96)	-20.30 (-37.33 to -3.26)	0.020*
CRP (mg/L)	21.16 (30.93)	93.92 ± 48.60	87.37 (71.36)	72.75 (53.04)	39.56 (35.72)	114.03 ± 43.73	112.22 (110.26)	81.71 (88.22)	-17.10 (-35.44 to 1.24)	0.068
PT, s	12.80 (1.60)	13.00 (1.90)	12.25 (2.20)	12.10 (2.20)	12.80 (1.30)	13.00 (1.60)	12.50 (1.50)	12.00 (1.80)	-0.18 (-0.88 to 0.52)	0.609
APTT, s	39.60 (16.30)	30.10 (6.00)	27.90 (2.60)	27.10 (3.90)	39.30 (17.30)	28.60 (3.40)	27.15 (3.80)	25.85 (3.40)	1.26 (-1.36 to 3.89)	0.346
D-D (mg/L)	5.12 (4.29)	4.18 (4.41)	5.18 (7.54)	8.59 (10.35)	5.02 (18.59)	6.72 (12.43)	6.51 (12.32)	7.42 (15.25)	-3.10 (-6.81 to 0.61)	0.101
Cr (μmol/L)	89.50 (54.20)	128.00 (77.00)	121.60 (119.50)	114.00 (82.80)	83.30 (30.20)	120.00 (72.50)	107.70 (101.50)	119.98 (79.50)	18.66 (-7.77 to 45.10)	0.166
BUN (mmol/L)	7.96 (3.32)	13.67 ± 5.63	15.30 ± 6.37	12.05 (7.94)	7.11 (2.07)	11.69 ± 3.72	12.65 ± 4.74	11.22 (3.99)	2.07 (0.41 to 3.74)	0.014*
AST (U/L)	50.80 (44.70)	59.00 (100.05)	52.30 (97.10)	65.20 (89.20)	44.10 (28.80)	47.60 (48.20)	46.80 (53.45)	49.85 (80.82)	42.28(-346.96 to 431.51)	0.831
ALT (U/L)	22.10 (33.20)	24.30 (36.80)	33.50 (72.50)	34.90 (67.40)	19.10 (23.80)	25.00 (36.50)	34.90 (43.10)	42.90 (68.20)	-14.60 (246.72 to 217.52)	0.902
NLR	15.80 (13.32)	17.23 (19.70)	22.60 (20.79)	22.15 (12.69)	18.20 (13.27)	24.75 (14.19)	23.33 (17.88)	19.43 (16.12)	-2.58 (-7.87 to 2.71)	0.339
SII	1875.01 (1949.31)	1881.00 (2188.05)	1689.60 (2088.83)	1664.88 (1976.87)	1908.00 (1686.09)	2326.50 (1857.99)	2108.00 (1905.71)	1691.00 (2101.10)	-221.23 (-837.19 to 394.72)	0.481
SIRI	11.40 (12.47)	13.94 (20.14)	14.67 (16.10)	13.28 (11.86)	11.65 (12.33)	15.92 (9.07)	15.45 (14.08)	10.98 (12.70)	-0.33 (-4.77 to 4.11)	0.885
SCI	24.63 (27.02)	21.18 (18.19)	25.44 (20.80)	26.70 (21.79)	27.73 (36.00)	26.37 (21.86)	30.88 (25.39)	39.45 (28.58)	-7.96 (-15.52 to -0.40)	0.039*

*: $P < 0.05$; NLR: Neutrophil lymphocyte ratio; Normal reference values are in parentheses in the first column

Secondary outcomes

Significant differences were observed between the two groups in terms of in-hospital mortality and stroke incidence, with the HA group showing lower rates of in-hospital death and stroke (Table 3). However, no significant differences were found in ventilator time, ICU stay duration, hospital stay duration, or hospitalization cost between the two groups. The 60-day postoperative survival of all patients was analyzed using Kaplan–Meier survival analysis (Fig. 2). The log-rank (Mantel-Cox) test was used to assess the differences in survival time between the two groups. The 60-day postoperative survival rate was significantly higher in the HA group compared to the control group ($P = 0.024 < 0.05$). Binary logistic regression was used to investigate the relationship between interventions, confounders, and outcomes such as in-hospital mortality and stroke incidence. When confounders were excluded, in-hospital mortality and stroke incidence were strongly associated with the use of HA380. However, When CPB duration and circulatory

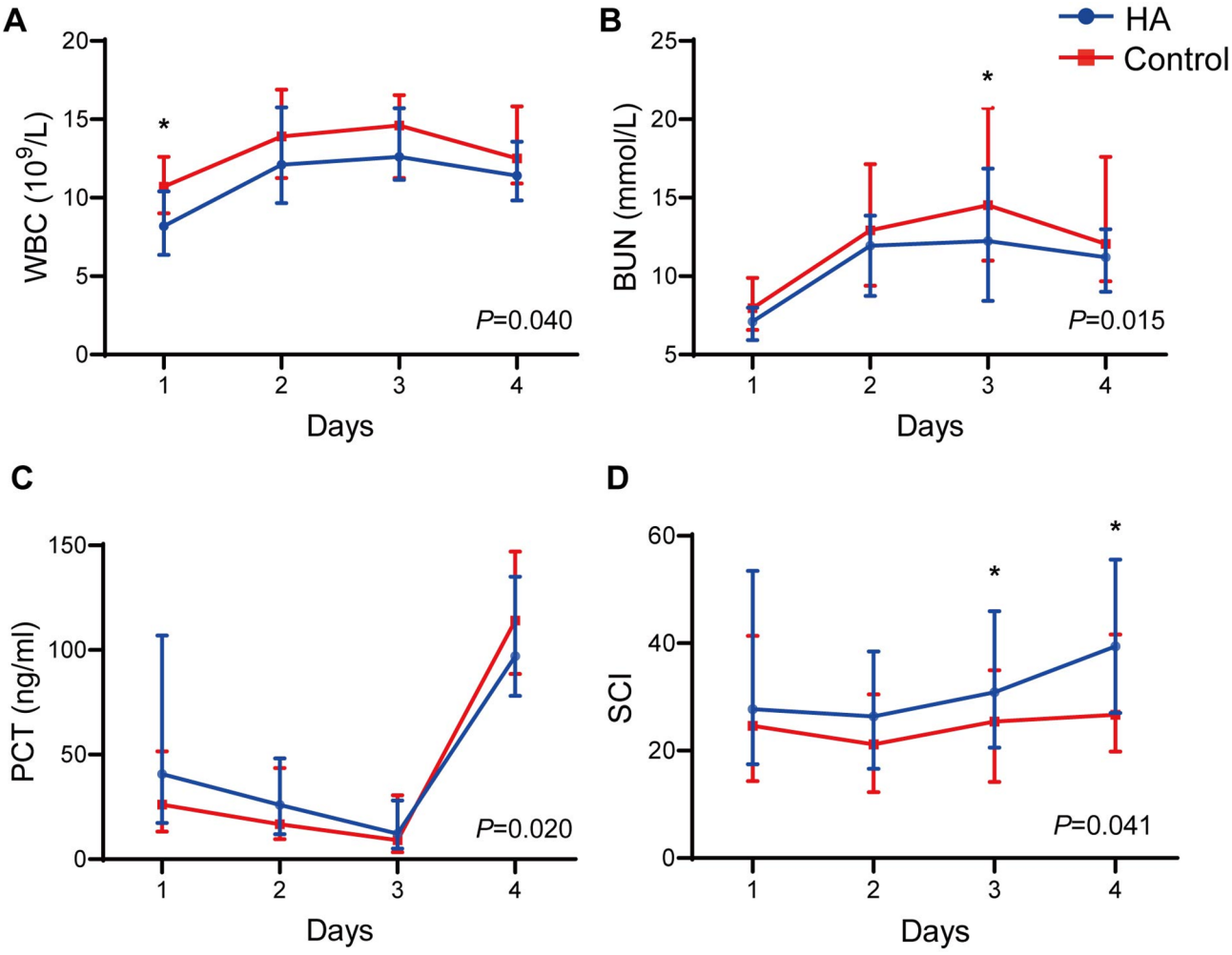

Fig. 1 The trends in WBC, BUN, PCT, and SCI over time. *: $p < 0.05$ VS Control group

Table 3 Comparison of secondary outcome in the HA group and control group

	Control ($n=45$)	HA ($n=45$)	*P*-value	95% CI
Ventilator time, min	75 (123)	74 (87)	0.524	(−33.43, 65.16)
ICU time, days	6 (6)	7 (7)	0.662	(−3.15, 2.01)
Length of stay, days	17 (11)	20 (8)	0.234	(−7.38, 1.83)
inpatient costs, ¥	270,636 (74344)	294,139 (86139)	0.426	(48714.19, 20764.37)
Hemodialysis	8 (17.78%)	11 (24.44%)	0.303	-
Stroke	19 (42.22%)	4 (8.89%)	0.001*	-
Mortality	16 (35.56%)	7 (15.56%)	0.026*	-

*: $P < 0.05$, ¥=RMB, 1RMB ≈ 0.13US dollar

arrest temperature were considered as confounders, in-hospital mortality was no longer significantly associated with intervention ($P = 0.443 > 0.05$); whereas the incidence of stroke remained significantly correlated with interventions ($P = 0.11 < 0.05$) (Table 4).

Discussion

Hemadsorption devices have been widely used in CPB and have shown remarkable efficacy in reducing intraoperative inflammatory factors, decreasing postoperative vasopressor requirements, and promoting postoperative recovery in patients [8, 20, 22]. However, not all patients can benefit from CPB [6]. In non-elective surgeries, especially emergency surgeries and infective endocarditis procedures, patients often present with high preoperative

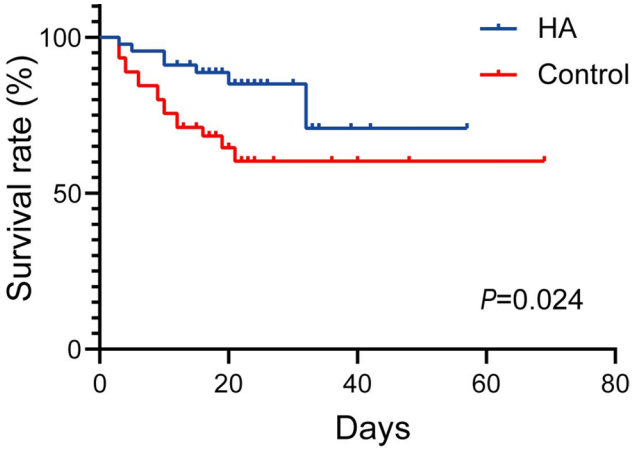

Fig. 2 The 60-day postoperative survival of all patients in two groups

inflammation levels. In such cases, intraoperative hemadsorption therapy has been shown to significantly reduce 30-day mortality and shorten ICU stays [10]. The HA380 cartridge, an extracorporeal blood adsorption device containing a porous resin, has been used utilized in CPB. However, no studies have yet demonstrated the therapeutic effect of HA380 during CPB in patients with ATAAD. As HA380 is known to adsorb a various cytokines produced during CPB, its potential benefits in this context are of interest. However, due to variations in treatment approaches, our hospital did not collect data on cytokines (IL-6/IL-8/IL-10, etc.), and thus this study cannot provide direct evidence of cytokine adsorption by HA380. Consequently, this article evaluates the impact of HA380 on patient prognosis solely through the analysis of postoperative inflammatory response and relevant clinical measures.

The baseline characteristics of patients show no significant differences between the HA and control groups. In terms of surgical procedure, the CPB time was shorter in the HA group compared to the control group, while the circulatory arrest temperature was higher. This may be attributed to the increasing skill and coordination of the surgical team, leading to reduced CPB times and higher temperatures during circulatory arrest. However, these factors may have affected the prognosis of patients in the HA group, potentially introducing bias into the study results. To address this, we used CPB time and circulatory arrest temperature as confounding factors when analyzing differences in primary outcome measures between the two groups. The WBC count was significantly reduced in the HA group compared to the control, which may be related to the adsorption of cytokines (IL-6/IL-8/IL-10, etc.) by the HA380 device. These inflammatory factors are known to promote inflammation and the activation and multiplication of immune cells [23, 24]. PCT, a marker significantly elevated in bacterial infections, is an important indicator of bacterial pneumonia and septicemia [25]. Postoperatively, PCT and SCI levels were higher in the HA group than in the control group. Different inflammation markers may represent different sources of inflammation, and postoperative inflammation in ATAAD patients can arise from various factors, including systemic blood circulation disorders, tissue and organ ischemia-reperfusion injury, extracorporeal circulation, surgical trauma, lung infection, and others. The anti-inflammation effect of the HA380 device require further investigation. No significant differences were observed in coagulation markers (AP, APTT, and D dimer) between the two groups. Among liver and kidney function measures, only BUN levels differed, with lower BUN levels in the HA group compared to the control group. For the secondary outcome measures, no significant differences were observed in hospital stay duration, ICU stay duration, ventilator use, or total hospitalizationl costs between the two groups. This suggests that the use of the HA380 device did not accelerate patient recovery or reduce hospitalization costs. However, in-hospital mortality and stroke incidence were lower in the HA group than in the control group. When CPB time and circulatory arrest temperature were considered as confounders, there was no significant correlation between in-hospital mortality and interventions, whereas stroke incidence was still significantly associated with interventions. Postoperative stroke is linked to higher in-hospital mortality, increased postoperative complications, and prolonged hospital stays [26, 27]. Nevertheless the specific mechanism underlying the reduction in stroke incidence and lower postoperative mortality require further study.

In summary, the use of the HA380 device during CPB did not reduce all kinds of postoperative inflammatory indicators in ATAAD surgery, and the reduction in inflammatory response was not obvious. However, the incidence of postoperative stroke reduced in HA group, which may contribute to improve prognosis, The specific mechanism underlying this improvement needs to be requires further investigation.

Table 4 Correlations of in-hospital mortality and stroke incidence with intervention measures

	Before correction			After correction		
	P-value	Exp (*B*)	95% CI	*P*-value	Exp (*B*)	95% CI
Mortality	0.033*	0.334	0.121–0.918	0.443	0.633	0.196–2.040
Stroke	0.001*	0.134	0.041–0.437	0.011*	0.188	0.052–0.680

*: $P < 0.05$

This study has several limitations. First, it was not a randomized controlled trial. Although propensity score matching was used, some confounding variables may still have influenced the outcomes. Second, the data came from were collected from a single center, which may limit the generalizability of the results and introduce randomness. Third, because interleukins are not routinely assayed after surgery, we lacked data on interleukins. Fourth, Preoperative brain malperfusion is a critical factor that could influence postoperative stroke rates. In our study, we did not systematically collect data on preoperative brain malperfusion, which is a limitation of our work. Finally, no significant differences were observed in postoperative coagulation indices or platelet counts in our study. Therefore, the mechanism by which the HA380 device affects the incidence of postoperative stroke in patient needs to be further explored.

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Author contributions
SG and ZW wrote the main manuscript. YZ and YG prepared the data collection. SG, ZW, YZ and YG prepared figures and tables. HZ and JL analyse and interpret of results. All authors reviewed the results and approved the final version of the manuscript. All authors would be informed each step of manuscript processing including submission, revision, revision reminder, etc.

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Data availability
The datasets used and analysed during the current study available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethics Committee of The First Hospital of Hebei Medical University [2023] Research Review No. (S00247). Informed consent was obtained from all the participants. All methods were carried out in accordance with Declaration of Helsinki.

Consent for publication
All the authors confirming that WRITTEN INFORMED consent was obtained from all subjects and/or their legal guardian(s).

Competing interests
The authors declare no competing interests.

Clinical trial number
Not Applicable.

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