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Article

The Effect of Intraoperative Hemoadsorption Therapy on Inflammatory Cytokines and Outcomes in Patients With Open Thoracoabdominal Aortic Aneurysm Repair

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Abstract

Background: To evaluate the effect of intraoperative hemoadsorption therapy on inflammatory cytokines and clinical outcomes in patients undergoing open thoracoabdominal aortic aneurysm (TAAA) repair. **Methods:** A total of 90 patients who underwent TAAA repair at Beijing Anzhen Hospital from May 2018 to December 2024 were enrolled and divided into a hemoadsorption group (HA group, n = 45) and a control group (n = 45). For patients in the HA group, an HA380 hemoadsorption cartridge was integrated in parallel into the cardiopulmonary bypass (CPB) circuit at the start of CPB. Patients in the control group underwent standard surgery without hemoadsorption and were matched to the HA group based on age (±5 years), sex, Crawford types, and comorbidities. We collected data on patient demographics, intraoperative variables (CPB duration, operative time, red blood cell transfusion volume, lactate levels), postoperative variables (duration of mechanical ventilation, intensive care unit [ICU] stay, hospital stay), inflammatory markers (interleukin-6, C-reactive protein, procalcitonin), and postoperative outcomes (mortality and complications). **Results:** No significant differences were observed between the two groups in CPB time or operative time ($p > 0.05$). Patients in the HA group had significantly shorter durations of mechanical ventilation, ICU stay, and hospital stay compared to those in the control group ($p < 0.05$). Intraoperative red blood cell transfusion volume and blood lactate levels were significantly lower in the HA group than those in the control group ($p < 0.05$). Similarly, serum levels of interleukin-6, C-reactive protein, and procalcitonin were significantly lower in the HA group than those in the control group at 24 and 48 hours ($p < 0.05$). There were no statistically significant differences in all-cause mortality or overall complication rates between the two groups ($p > 0.05$). However, the incidence of acute kidney injury (AKI) was significantly lower in the HA group (37.78%) than in the control group (64.44%, $p < 0.05$), representing a 41.37% relative reduction. **Conclusion:** In TAAA repair, intraoperative hemoadsorption was associated with a significantly lower incidence of AKI, lower serum levels of interleukin-6, C-reactive protein, and procalcitonin at 24 and 48 hours, and shorter durations of mechanical ventilation, ICU stay, and hospital stay compared with the control group. All-cause mortality and rates of other complications were similar between the two groups.

Keywords: thoracoabdominal aortic aneurysm; hemoadsorption; inflammatory cytokines; acute kidney injury; cardiopulmonary bypass; complications

1. Introduction

Open thoracoabdominal aortic aneurysm (TAAA) repair remains a primary intervention for TAAA and aortic dissection. However, the extensive surgical trauma, prolonged cardiopulmonary bypass (CPB) [1], and contact of blood with the artificial surfaces of the CPB circuit can trigger inflammatory pathways, involving monocytes and macrophages. This promotes the release of inflammatory cytokines and can lead to a systemic inflammatory response syndrome (SIRS) [2], which is often associated with multi-organ dysfunction and postoperative mortality rates ranging from 5% to 25% [3,4,5]. Extracorporeal hemoadsorption therapy has emerged as a potential intervention to mitigate these effects. This therapy may attenuate inflammation-induced organ damage by removing inflammatory mediators—including interleukin-6 (IL-6), C-reactive protein (CRP), procalcitonin (PCT), and tumor necrosis factor-α (TNF-α)—as well as other toxic substances from the circulation. As a result, it has shown promise in improving outcomes in various clinical settings, including severe infections (e.g., sepsis and septic shock) and complex cardiac and major vascular surgeries.

Currently, two hemoadsorption devices are widely used: CytoSorb® (CytoSorbents Inc., Princeton, NJ, USA) composed of porous polyethylene microspheres (300–800 µm) with a surface area of 45,000 m^2 per canister, and adsorbs molecules weighing 10–50 kDa, and HA380 hemoperfusion cartridge (Jafron Biomedical Co., Ltd., Zhuhai, China) consists of styrene-divinylbenzene copolymer microspheres (450 µm) with a surface area of 50,000 m^2 per

cartridge, and is capable of adsorbing molecules weighing 5–60 kDa (A comparison of the two devices is provided in the **Supplementary Material**) [6,7]. Both devices demonstrate similar biocompatibility and clinical indications, theoretically disrupting inflammatory cascades by adsorbing mediators (e.g., IL-6, CRP, PCT, TNF-α) and toxins (including drugs) [8,9]. Although studies suggest clinical benefits in complex cardiac and major vascular surgeries and critical care settings (e.g., sepsis, septic shock) [10,11], the efficacy of these devices remains controversial. For instance, Jerman et al. [12] observed no significant differences in lactate, CRP, or IL-6 levels between 52 critically ill patients treated with CytoSorb® and 94 controls, and a meta-analysis of randomized trials even indicated that CytoSorb® might increase mortality and adverse events in critically ill patients with inflammatory conditions [13].

Given the limited evidence on the clinical benefits of the HA380 hemoadsorption cartridge specifically in TAAA surgery, we conducted a 1:1 matched case-control study at Beijing Anzhen Hospital, Capital Medical University, China. The study compared 45 TAAA patients treated with the HA380 hemoperfusion cartridge with 45 matched controls, to evaluate its impact on postoperative complications and inflammatory cytokine levels. Our findings may provide valuable insights for optimizing TAAA surgical management.

2. Materials and Methods

2.1 Patient Selection and Grouping

The retrospective matched case-control study was approved by the Ethics Committee of Beijing Anzhen Hospital (Anzhen EC 2026159x). Written informed consent was obtained from all included patients and their families.

A total of 90 patients who underwent open TAAA repair with CPB support at Beijing Anzhen Hospital, Capital Medical University, between May 2018 and December 2024 were enrolled. They were divided into a hemoadsorption group (HA group, n = 45) and a control group (n = 45). Allocation to the HA group was based on the intraoperative use of an HA380 hemoperfusion cartridge in the CPB circuit, which was implemented according to the patient's financial considerations and the surgeon's discretion.

The control group was matched 1:1 with the HA group for age (±5 years), sex, Crawford classification, and comorbidities. Baseline characteristics showed no significant differences between the two groups ($p > 0.05$).

Inclusion criteria were: (1) age 18–70 years; (2) first-time TAAA repair; and (3) elective or emergency surgery.

Exclusion criteria were: (1) pregnancy; (2) age <18 years; (3) active malignancy; (4) systemic or local infections; (5) hematologic disorders; (6) heart failure (NYHA Class III/IV); and (7) hepatic or renal dysfunction (defined as alanine aminotransferase [ALT] or serum creatinine levels above the upper limit of normal).

2.2 Surgical Procedure

Combined intravenous-inhalation anesthesia with double-lumen endotracheal intubation was administered. A lumbar puncture was performed to place a cerebrospinal fluid (CSF) drainage tube. The drainage rate was maintained at 10–15 mL/h, and CSF pressure was monitored and kept at ≤10 mmHg (1 mmHg = 0.133 kPa). Continuous CSF drainage was maintained for 48 hours postoperatively. Left radial artery and dorsalis pedis artery catheterization was performed to monitor upper and lower limb blood pressure. An S5 extracorporeal circulation machine and an adult membrane oxygenator were used. Standard CPB circuit priming was performed using 1000 mL of succinylated gelatin solution and 500 mL of compound electrolyte solution, supplemented with heparin (1 mg/kg). A left thoracoabdominal incision was made, typically through the 6th to 9th intercostal space. A Fr26 catheter was inserted into the inferior vena cava via the femoral vein, and a Fr21 or Fr24 arterial catheter was inserted into the femoral or iliac artery to establish CPB. Mild hypothermia (approximately 34 °C) was maintained for spinal cord protection and to reduce the risk of ventricular fibrillation. In the HA group, upon initiation of CPB, an HA380 hemoperfusion cartridge was connected to the CPB system for hemoperfusion therapy. Venous return flow was adjusted to maintain upper limb arterial pressure between 90 mmHg and 120 mmHg and distal pressure between 60 mmHg and 80 mmHg. The aorta and visceral arteries were fully exposed. After full exposure of the aorta and visceral arteries, the normal aortic segments proximal and distal to the aneurysm were dissected, and branch vessels were managed accordingly. Normal aortic segments proximal and distal to the aneurysm were dissected, and branch vessels were managed. The proximal aorta, visceral arteries, and distal aorta were sequentially clamped. The diseased aortic segment was replaced with a synthetic graft, and the visceral arteries (celiac trunk, superior mesenteric artery, and renal arteries) were rapidly anastomosed to the graft before discontinuing CPB. Following reperfusion, meticulous hemostasis was achieved, and perfusion to visceral organs was assessed. Postoperative monitoring focused on spinal cord function, renal function, and visceral organ perfusion, and any complications were actively managed.

2.3 Perioperative Management Protocol

The perioperative management protocol included the following key elements:

(1) Weaning from CPB: This was initiated after the patient's body temperature returned to 36 °C, with achievement of hemodynamic stability (MAP >65 mmHg), normal blood gas analysis results, and adequate cardiac filling.

(2) Blood transfusion indication: Transfusion was indicated when hemoglobin levels dropped below 8 g/dL.

(3) Data collection: Blood samples were collected at 24 and 48 hours postoperatively for cytokine measurement.

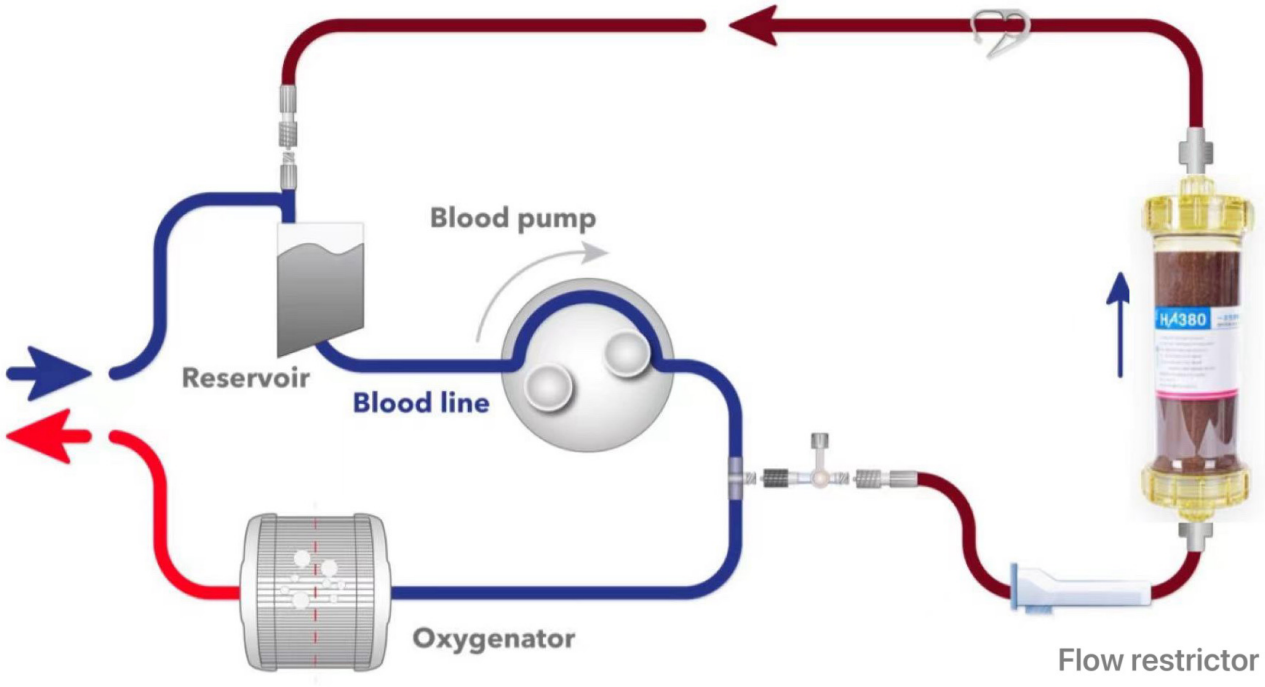

Fig. 1. Schematic diagram of HA380 hemoperfusion cartridge integrated into the CPB circuit (blood flow path: arterial line → HA380 hemoperfusion cartridge → reservoir → pump → patient) [14]. The blue arrow indicates the direction of venous blood entering the extracorporeal circulation system; the purple arrow indicates the blood flowing through the HA380; the red arrow indicates oxygenated and purified blood flowing back into the arterial system.

(4) Weaning from mechanical ventilation: The process was initiated when the following criteria were met: adequate control of the underlying disease, clear consciousness, muscle strength ≥ grade 2, good cough reflex, successful spontaneous breathing trial (SBT) without respiratory distress or increased cardiac load, and SpO_2 ≥90%.

2.4 Application of the HA380 Cartridge

The HA380 hemoperfusion cartridge was connected in parallel with the CPB circuit and remained in use until CPB was discontinued (Fig. 1, Ref. [14]). Within the circuit, blood flowed through the HA380 cartridge, where inflammatory cytokines and toxins were adsorbed and removed. The purified blood was then returned to the reservoir and recirculated to the patient via the blood pump.

2.5 Definition of Primary Outcome Variables

Patient demographic data (sex, age, and body mass index [BMI]), comorbidities, routine blood test results, blood biochemical indicators, liver and kidney function parameters, estimated glomerular filtration rate (eGFR), Crawford classification, and Marfan syndrome were recorded.

The primary outcome measures included: (1) Surgical metrics: duration of CPB and total operation time; (2) Intraoperative measures: volume of red blood cells transfused and intraoperative blood lactate levels; (3) Postoperative inflammatory markers: serum levels of IL-6, CRP, and PCT at 24 and 48 hours post-surgery; (4) Safety outcomes: in-hospital and 30-day all-cause mortality, and the incidence of major adverse events, including acute kidney injury (AKI), spinal cord injury (SCI), and other complications (see **Supplementary Table 2** in the **Supplementary Data**).

AKI was defined as an absolute increase in serum creatinine of ≥26 μmol/L within 48 hours or an increase to ≥150% of baseline within 7 days of surgery according to the KDIGO clinical practice guidelines [15,16].

Staging of AKI was defined according to the KDIGO criteria [15,16] as follows:

Stage 1: Increase in serum creatinine to 1.5–1.9 times baseline, or an increase of ≥0.3 mg/dL (≥26.5 μmol/L), or urine output <0.5 mL/kg/h for 6–12 hours.

Stage 2: Increase in serum creatinine to 2.0–2.9 times baseline, or urine output <0.5 mL/kg/h for ≥12 hours.

Stage 3: Increase in serum creatinine to 3.0 times baseline, or increase to ≥4.0 mg/dL (≥353.6 μmol/L), or initiation of renal replacement therapy, or urine output <0.3 mL/kg/h for ≥24 hours, or anuria for ≥12 hours. For patients <18 years, a decrease in eGFR to <35 mL/min per 1.73 m^2 also constitutes Stage 3.

SCI was defined as paraparesis or paraplegia confirmed by a neurologist.

2.6 Safety Evaluation

In addition to complications and mortality, safety indicators included white blood cell (WBC) count, neutrophil

Table 1. Baseline characteristics of the patients in the two groups (n = 45/group).

Variables	Control group	HA group	X^2/t	*p*-value
Age, (years)	42.82 ± 13.22	42.16 ± 12.06	0.250	0.803
Male, n (%)	36 (80.00)	34 (75.56)	0.257	0.800
Crawford type1, n (%)	0 (0.00)	2 (4.44)	2.045	0.494
Crawford type 2, n (%)	14 (31.11)	9 (20.00)	1.460	0.227
Crawford type 3, n (%)	29 (64.44)	28 (62.22)	0.048	0.827
Crawford type 4, n (%)	0 (0.00)	2 (4.44)	2.045	0.494
Crawford type 5, n (%)	2 (4.44)	4 (8.89)	0.714	0.677
Renal A involvement, n (%)	27 (60.00)	24 (53.33)	0.407	0.671
Marfan Syndrome, n (%)	17 (37.78)	16 (35.56)	0.048	1.000

Notes: A, artery; Renal A involvement, Renal artery involvement indicates that the renal arteries were affected by the TAAA lesion.

count, platelet count, creatinine level, uric acid level, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and electrolyte levels.

2.7 Statistical Analysis

All collected data were imported into the SPSS database and analyzed using SPSS 25.0 (IBM Corporation, Armonk, NY, USA). Categorical data were expressed as percentages (%), and intergroup comparisons were performed using the chi-square test. Continuous data were tested for normal distribution. Normally distributed data were presented as mean ± standard deviation (mean ± SD), and intergroup comparisons were conducted using independent samples *t*-tests. A two-tailed test was applied with a significance level of $\alpha = 0.05$, and $p < 0.05$ was considered statistically significant.

3. Results

3.1 Baseline Characteristics

There were no significant differences in age, sex, Crawford classification, comorbidities, body mass index (BMI), smoking history, renal function, or other baseline characteristics between the two groups ($p > 0.05$; See Table 1 and **Supplementary Table 1** in the **Supplementary data**). These findings indicate that the patient characteristics were well-balanced and comparable.

3.2 Postoperative Outcomes

Intergroup comparisons of postoperative outcomes are presented in Table 2. No significant differences were observed between the two groups in duration of surgery, CPB time, or intraoperative blood loss volume ($p > 0.05$). However, the HA380 group demonstrated significantly lower values than the control group for red blood cell transfusion volume (1.60 ± 2.20 U vs. 2.82 ± 3.36 U; $p = 0.044$) and peak lactate levels (intraoperative: 4.13 ± 1.62 vs. 5.52 ± 3.17 mmol/L, $p = 0.010$; postoperative: 2.92 ± 1.05 vs. 4.28 ± 4.14 mmol/L, $p = 0.041$). Additionally, the HA group showed significantly lower levels of inflammatory markers (IL-6, CRP, and PCT) at both 24 hours and 48 hours postoperatively compared to the control group ($p < 0.05$). Although no significant differences were observed in mortality rates or major adverse events ($p > 0.05$), the HA group had a significantly lower incidence of AKI (40.00% vs. 64.44%, $p < 0.05$) compared with the control group. However, in the subgroup analysis of AKI staging and cases requiring continuous renal replacement therapy (CRRT), there were no significant differences between groups ($p > 0.05$), a finding attributed to the insufficient sample size. The rates of other postoperative complications were not statistically different between the two groups ($p > 0.05$; see **Supplementary Table 2**). Notably, the HA group experienced significantly shorter hospital stays (26.36 ± 5.15 vs. 30.76 ± 9.54 days; $p = 0.008$), ventilation time (27.42 ± 17.09 vs. 40.09 ± 27.98 hours; $p = 0.011$), and intensive care unit (ICU) stays (40.16 ± 22.89 vs. 54.36 ± 28.57 hours; $p = 0.011$).

3.3 Safety Profile

There were no significant differences in WBC, creatinine, ALT, AST, and electrolytes in the two groups after the surgery ($p > 0.05$; See Table 3).

4. Discussion

Open TAAA repair is a highly invasive procedure associated with substantial bleeding risks, prolonged CPB time, and a pronounced systemic inflammatory response [17]. During CPB, contact of blood with artificial surfaces of the circuit triggers the release of inflammatory mediators, potentially leading to a cytokine storm and multiorgan complications [10,18,19]. Therefore, mitigating these perioperative inflammatory reactions is crucial for improving patient outcomes.

The HA380 hemoperfusion device is an extracorporeal blood purification system [20] that consists of neutral macroporous resin beads. These are polymer microspheres with a porous structure and an exceptionally high

Table 2. Patient outcomes after surgery.

Variables	Control group	HA group	X^2/t	*p*-value
Duration of surgery, (min)	588.89 ± 76.20	587.02 ± 104.19	0.097	0.923
Duration of CPB, (min)	122.31 ± 35.29	117.80 ± 45.98	0.522	0.603
Blood loss volume, (mL)	2043.20 ± 1367.06	1628.89 ± 805.56	1.761	0.082
RBC transfusion volume, (U)	2.82 ± 3.36	1.60 ± 2.20	2.045	0.044
Peak lactate level 1, (mmol/L)	5.52 ± 3.17	4.13 ± 1.62	2.620	0.010
Peak lactate level 2, (mmol/L)	4.28 ± 4.14	2.92 ± 1.05	2.070	0.041
IL-6 at post-operative 24 h, (ng/L)	142.27 ± 63.02	85.13 ± 55.95	4.549	0.000
IL-6 at post-operative 48 h, (ng/L)	84.12 ± 61.29	56.57 ± 38.96	2.503	0.014
CRP at post-surgery 24 h, (mg/L)	82.71 ± 38.39	58.84 ± 22.97	3.579	0.000
CRP at post-surgery 48 h, (mg/L)	87.56 ± 49.01	55.38 ± 38.96	3.449	0.001
PCT at post-surgery 24 h, (ng/L)	17.17 ± 11.10	12.64 ± 6.77	2.339	0.022
PCT at post-surgery 48 h, (ng/L)	14.11 ± 8.96	9.85 ± 7.83	2.401	0.018
AKI at post-surgery 48 h, n (%)	29 (64.44)	17 (37.78)	6.403	0.011
AKI Stage 1, n (%)	16 (35.56)	11 (24.44)	1.323	0.250
AKI Stage 2, n (%)	8 (17.78)	4 (8.89)	1.538	0.215
AKI Stage 3, n (%)	5 (11.11)	2 (4.44)	1.394	0.238
CRRT, n (%)	5 (11.11)	2 (4.44)	1.394	0.238
Spinal cord injury, n (%)	7 (15.56)	6 (13.33)	0.090	1.000
Duration of hospital stay, (D)	30.76 ± 9.54	26.36 ± 5.15	2.723	0.008
Duration of ventilator, (h)	40.09 ± 27.98	27.42 ± 17.09	2.519	0.011
Duration of ICU, (h)	54.36 ± 28.57	40.16 ± 22.89	2.602	0.011
In-hospital all-cause mortality, n (%)	1 (2.22)	1 (2.22)	0.000	1.000
30-day all-cause mortality, n (%)	4 (8.89)	2 (4.44)	0.714	0.398
Major adverse events, n (%)	39 (86.67)	40 (88.89)	0.104	0.784

Note: AKI, acute kidney injury; CRP, C-reactive protein; CRRT, continuous renal replacement therapy; D, day; h, hours; ICU, Intensive care unit; IL-6, interleukin-6; PCT, procalcitonin; Peak lactate level 1, intraoperative peak lactate level; Peak lactate level 2, postoperative peak lactate level; RBC, Red blood cells.

Table 3. Comparison of blood safety measures on the postoperative day.

Variables	Control group	HA group	X^2/t	*p*-value
WBC (10^9/L)	11.94 ± 4.62	13.32 ± 4.82	1.376	0.172
Neutrophil (10^9/L)	10.29 ± 4.44	11.66 ± 4.94	1.357	0.178
Lymphocyte (10^9/L)	0.85 ± 0.42	0.86 ± 0.49	0.083	0.934
Platelet (10^9/L)	75.82 ± 38.63	84.20 ± 33.26	1.102	0.274
ALT, (U/L)	33.69 ± 6.16	35.60 ± 6.51	1.431	0.156
AST, (U/L)	37.76 ± 4.27	36.60 ± 4.16	1.300	0.197
Creatinine, (μmol/L)	123.37 ± 50.87	116.10 ± 57.44	0.636	0.527
Uric acid, (μmol/L)	324.53 ± 100.59	342.84 ± 80.58	0.953	0.343
eGFR, (mL/min/1.73 m^2)	70.32 ± 32.13	76.55 ± 32.01	0.921	0.360
Creatine kinase, (U)	2717.00 ± 871.49	2660.95 ± 788.85	0.320	0.750
Sodium ion, (mmol/L)	148.49 ± 4.59	147.89 ± 3.71	0.682	0.497
Potassium ion, (mmol/L)	4.43 ± 0.55	4.40 ± 0.47	0.311	0.757

Note: ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; Creatinine, Serum creatinine values measured at 48 hours after surgery; eGFR, Estimated Glomerular Filtration Rate; WBC, White blood cells.

surface area (typically >1000 m^2/g), enabling the adsorption of molecules weighing 10–60 kDa and the direct removal of inflammatory mediators, toxins, and metabolic waste products from the circulation.

Studies have shown that the use of HA380 is associated with stabilized hemodynamics, reduced end-organ damage, and a lower rate of postoperative complications [21,22], effects likely attributable to the attenuation of cytokine storms [23].

Key advantages of the HA380 include rapid adsorption kinetics, high adsorptive capacity, excellent biocompatibility, and a proven safety profile. It does not increase hemolysis, leukocyte or platelet disruption, or other adverse events related to cardiac surgery [24], and may further enhance microcirculatory perfusion while minimizing endothelial injury [25,26].

In this 1:1 matched case-control study, red blood cell transfusion requirements were significantly lower in the HA380 group (1.60 ± 2.20 U) than in the control group (2.82 ± 3.36 U, $p < 0.05$). Both intraoperative (4.13 ± 1.62 mmol/L vs. 5.52 ± 3.17 mmol/L) and postoperative (2.92 ± 1.05 mmol/L vs. 4.28 ± 4.14 mmol/L, $p < 0.05$) peak lactate levels were also lower in the HA380 group compared with the control group. Furthermore, postoperative inflammatory markers (IL-6, CRP, and PCT) were significantly reduced in the HA380 group at both 24 and 48 hours (all $p < 0.05$), confirming the device's efficacy in clearing inflammatory mediators. The role of these mediators in renal injury is well-established. Interleukin-6 (IL-6) directly induces apoptosis in renal tubular epithelial cells via the gp130 receptor signaling pathway, increases renal vascular permeability, and consequently leads to a decline in renal function. Research indicates that IL-6 also promotes the progression from AKI to renal fibrosis through mechanisms such as DNA methylation [27]. TNF-α binds to its receptor to initiate the caspase pathway, inducing apoptosis and necrosis in renal tubular cells. Additionally, TNF-α activates the nuclear factor kappa-B (NF-κB) pathway, stimulating the release of IL-1β, IL-6, IL-8, and chemokines, which in turn induce the infiltration of neutrophils and macrophages into the kidney, resulting in renal tissue damage. TNF-α further exacerbates injury by inducing vasoconstriction and promoting microvascular thrombosis, which reduces renal blood flow [28]. Procalcitonin (PCT) serves as an indicator of inflammatory storm intensity. Elevated PCT levels signify uncontrolled systemic inflammation, a condition that can lead to persistent hypotension, reduced renal perfusion, and microcirculatory disturbances, thereby triggering or worsening AKI [29]. Therefore, the observed reduction in AKI incidence in the HA380 group (40.00% vs. 64.44%, $p < 0.05$) is likely attributable to the effective adsorption of lactate and these key cytokines [2]. The renal protective effects of cytokine reduction are mediated by several key mechanisms:

(1) **Interruption of the inflammatory cascade**. By removing large quantities of cytokines, hemoadsorption can break the positive feedback loop of the "cytokine storm", thereby attenuating the systemic inflammatory insult to the kidneys.

(2) **Reduction of renal tubular epithelial cell death**. Cytokines, particularly TNF-α and IL-6, bind directly to receptors on renal tubular cells, triggering intracellular death signaling pathways. Lowering circulating cytokine levels reduces this receptor activation, helping to preserve mitochondrial function and cytoskeletal integrity, thus slowing or preventing tubular cell apoptosis and necrosis.

(3) **Improvement of renal hemodynamics and microcirculation**. High levels of inflammatory mediators like TNF-α induce renal vasoconstriction, endothelial dysfunction, and capillary leakage. Their removal helps restore vasodilatory capacity, increase renal blood flow, and reduce endothelial damage and vascular permeability. This, in turn, mitigates renal interstitial edema and improves renal oxygen delivery.

(4) **Attenuation of leukocyte infiltration and oxidative stress**. Cytokines act as chemoattractants, recruiting neutrophils and macrophages to the kidneys. Lowering cytokine levels reduces this inflammatory cell infiltration into the renal tissue interstitium, thereby decreasing the production of reactive oxygen species and limiting oxidative stress-induced damage [30,31]. In summary, lowering cytokines like IL-6, TNF, and PCT helps protect kidney function by reducing toxic cell damage, improving blood flow, and blocking the pathway that leads to inflammatory cell buildup.

Our findings are consistent with previous research. Condello et al. [32] observed a lower rate of AKI with HA380 use in elective CPB patients (10% vs. 40%, $p = 0.027$), while Wang et al. [9] reported that hemoperfusion was linked to attenuated IL-6 levels and a reduced incidence of AKI (25.4% vs. 44.6%, $p = 0.032$) in patients with aortic dissection. Similarly, Mehta et al. [33] found that the use of HA380 hemoperfusion in aortic root surgery was associated with reduced vasopressor requirements, shortened ICU and hospital stays, and decreased IL-6 and PCT levels ($p < 0.05$). Crucially, safety outcomes—including mortality, complications, and white blood cell counts—did not differ between groups ($p > 0.05$) in our study, further supporting the safety profile of HA380 [34]. Systematic reviews reported by Matejic-Spasic et al. [35] (n = 1057 across 29 studies) showed that hemodynamic stability and the absence of adverse events were associated with using HA 380.

The results of this study and those of previous studies indicated that hemoadsorption could theoretically and practically clear specific molecules, such as endotoxin, as well as non-specifically adsorb pro-inflammatory mediators. However, despite decades of study, the results of hemoadsorption therapy have been inconsistent [36]. For instance, a systematic review and meta-analysis by Becker et al. [37] on CytoSorb® therapy found no significant differences in ICU length of stay, lactate, or IL-6 levels. A subsequent randomized controlled trial [38] reported that HA therapy did not reduce IL-6 levels upon ICU admission and found no intergroup differences in other cytokine concentrations, organ dysfunction, or clinical outcomes. In a study of 146 critically ill patients, Jerman et al. [12] similarly found no significant differences in IL-6, lactate, CRP, PCT levels, or mortality. More concerning, a systematic review with meta-analysis by Heymann et al. [13] suggested

that CytoSorb® therapy might be associated with increased mortality and adverse events in critically ill patients with inflammatory conditions, although another systematic review by Saldaña-Gastulo et al. [39] found no significant effect on mortality. These conflicting results may reflect critical differences in patient selection. In cardiac surgery, CPB induces a relatively predictable and uniform inflammatory cascade, potentially making targeted adsorption more effective. In contrast, ICU patients with sepsis present with multifactorial and highly variable pathologies, where numerous confounders may diminish the observable efficacy of HA therapy. Current evidence therefore supports the use of HA380 in complex surgeries involving CPB [40], whereas its utility in sepsis or septic shock requires further validation through rigorous, prospective trials [41]. Selective application in settings with a high and predictable inflammatory burden, such as during CPB, may be the key to optimizing patient outcomes with this technology.

5. Study Limitations

This study has several limitations. First, the small sample size may limit the generalizability of the findings. Second, as a retrospective case-matched study with incomplete data (only IL-6, CRP, and PCT were measured; TNF-α was not assessed), it does not demonstrate whether the HA380 hemoadsorption device has comparable effects on other inflammatory markers, such as TNF-α. Therefore, the results require validation through larger multicenter randomized controlled trials. Residual confounding risks related to perioperative management, surgeon experience, anesthetic regimens, transfusion strategies, and ICU practices will be excluded in a prospective randomized controlled trial to be conducted in the future.

6. Conclusion

In TAAA repair, intraoperative hemoadsorption was associated with a significantly lower incidence of AKI, lower serum levels of interleukin-6, C-reactive protein, and procalcitonin at 24 and 48 hours, and shorter durations of mechanical ventilation, ICU stay, and hospital stay compared with the control group. All-cause mortality and rates of other complications were similar between the two groups.

Abbreviations

AKI, Acute Kidney Injury; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; BMI, Body Mass Index; CPB, Cardiopulmonary Bypass; CRP, C-reactive protein; D, Day; eGFR, Estimated Glomerular Filtration Rate; H, Hours; ICU, Intensive Care Unit; IL-6, Interleukin-6; kDa, kilo-Dalton; PCT, Procalcitonin; TAAA, Thoracoabdominal Aortic Aneurysm; TNF-α, Tumor Necrosis Factor-α; U, Units; WBC, White Blood Cells.

Availability of Data and Materials

All data generated or analyzed during this study are included in this article and its supplementary data. Further enquiries can be directed to the corresponding authors.

Author Contributions

MG, FH and TL performed the study design, participated in the surgical work, and wrote the main manuscript text and cooperated with other authors in clinical research work. KW, XY and LF had done the data collection, established the database and participated in the drafting of the article. MG and JX were responsible for data analysis, figures and tables. JX designed the research study, performed the research, analyzed the data and made critical revisions to the important content of the manuscript. All authors have read and approved the publication of this manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of Beijing Anzhen Hospital (ethics approval number: Anzhen EC 2026159x). The study was conducted according to the guidelines of the Declaration of Helsinki. All patients signed the informed consent form.

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Conflicts of Interest

The authors declare no conflicts of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/HSF52122.

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