

Prophylactic Low-Dose Dexamethasone for Postoperative Pulmonary Complications in Type B Aortic Dissection: A Retrospective Cohort Study

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AIM: To evaluate the impact of prophylactic low-dose glucocorticoids on the incidence of postoperative pulmonary complications (PPCs) and perioperative recovery parameters in patients undergoing surgery for type B aortic dissection.

METHODS: This retrospective cohort study analyzed 240 patients who underwent type B aortic dissection (TBAD) surgery at Taizhou Hospital of Zhejiang Province Affiliated to Wenzhou Medical University from January 2023 to December 2025. Patients were divided into an intervention group ($n = 118$) receiving a single dose of dexamethasone (5 mg intravenously) after anesthesia induction and a control group ($n = 122$) receiving standard care. To minimize selection bias, propensity score matching (PSM) was performed using 1:1 nearest neighbor matching with a caliper of 0.2 times the standard deviation of the propensity score. Covariates included age, sex, body mass index, diabetes, smoking history, preoperative C-reactive protein, preoperative $\text{PaO}_2/\text{FiO}_2$ ratio, operative time, intraoperative blood transfusion volume, and surgical procedure (thoracic endovascular aortic repair [TEVAR] vs. open surgery). After matching, 93 well-balanced pairs were obtained. The primary outcome was the incidence of PPCs within 30 days postoperatively. Secondary outcomes included the duration of mechanical ventilation, intensive care unit (ICU) and hospital length of stay, as well as postoperative inflammatory markers (C-reactive protein [CRP], white blood cell [WBC]) and oxygenation ($\text{PaO}_2/\text{FiO}_2$ ratio) at 24 hours.

RESULTS: After PSM, baseline characteristics were well balanced between groups, with all standardized mean differences < 0.1 . The intervention group showed a significantly lower incidence of PPCs compared to the control group (20.43% vs. 34.41%, $p = 0.033$). The intervention was also associated with significantly shorter mechanical ventilation duration ($p = 0.008$), ICU stay ($p = 0.003$), and total hospital stay ($p = 0.009$). Additionally, the intervention group demonstrated significantly lower postoperative CRP and WBC levels and a higher $\text{PaO}_2/\text{FiO}_2$ ratio at 24 hours (all $p < 0.001$). No significant differences were observed in safety outcomes including new-onset infection, wound healing impairment, or hyperglycemia.

CONCLUSIONS: Prophylactic low-dose glucocorticoids were associated with a lower incidence of PPCs and improved perioperative recovery in patients undergoing TBAD surgery, including shorter ventilation time and the length of hospital stay, attenuated inflammatory response, and better oxygenation. These findings suggest potential clinical benefits with a favorable safety profile, but causality cannot be inferred due to the retrospective design. Therefore, prospective multicenter studies are warranted to validate these results.

Keywords: type B aortic dissection; dexamethasone; perioperative; inflammatory response; pulmonary complications; prophylactic application

Introduction

Type B aortic dissection (TBAD) represents a critical manifestation within the spectrum of acute aortic syndromes, characterized by an acute onset and complex clinical trajectory. Without timely intervention, it is associated with

exceedingly high mortality rates. Advances in thoracic endovascular aortic repair (TEVAR) and open surgical techniques have substantially improved survival outcomes for patients undergoing TBAD surgery. However, perioperative complications remain a significant impediment to favorable prognoses, with postoperative pulmonary complications (PPCs) being particularly prominent. Previous studies have indicated that the incidence of PPCs in patients undergoing aortic surgery ranges from 20% to 40%, including acute respiratory distress syndrome (ARDS), pneumonia, atelectasis, and pulmonary edema. These complications not only prolong durations of mechanical ventilation and intensive care unit (ICU) stay but also increase in-hospital mortality and healthcare costs [1,2].

The pathogenesis of PPCs is multifactorial, involving systemic inflammatory response syndrome (SIRS) triggered

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by surgical trauma, ischemia-reperfusion injury, fluid overload, increased pulmonary vascular permeability, and respiratory depression induced by anesthetic agents [3]. In TBAD surgery, particularly when involving thoracoabdominal aortic repair, prolonged operative duration, extensive surgical trauma, and frequent requirements for substantial blood transfusion, cardiopulmonary bypass, or deep hypothermic circulatory arrest further increase the risk of pulmonary injury [4]. Consequently, investigating effective perioperative interventions to mitigate inflammatory responses and preserve pulmonary function has emerged as a critical focus of clinical research.

Glucocorticoids, as potent anti-inflammatory agents, exert their effects by inhibiting signaling pathways such as nuclear factor-kappa B (NF- κ B), reducing the release of pro-inflammatory cytokines (e.g., tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6)), and decreasing vascular permeability. These mechanisms theoretically confer the potential to attenuate lung injury [5]. Within the realm of cardiac surgery, numerous randomized controlled trials (RCTs) and meta-analyses have demonstrated that a single perioperative dose of dexamethasone or methylprednisolone significantly reduces postoperative levels of inflammatory markers (C-reactive protein (CRP), IL-6). Some studies have further observed clinical benefits including shortened ICU stays and a reduced risk of acute respiratory failure [6,7,8]. Nevertheless, evidence regarding improvements in major complication endpoints (e.g., mortality, myocardial infarction) remains inconsistent, with considerable heterogeneity in efficacy outcomes [9].

In aortic dissection surgery, most studies have focused on type A dissection with conflicting results [10,11]. For TBAD, evidence is extremely limited. Wu et al. [12] reported that dexamethasone facilitated postoperative recovery of vital signs but did not affect mortality or infection in patients with chronic type B dissection post-TEVAR. To date, no high-quality RCTs exist for TBAD, and the heterogeneity observed in studies of type A aortic dissection suggests that patient selection, surgical technique, drug dosage, and timing are critical determinants of treatment response. The selection of an appropriate glucocorticoid dosage is crucial. High-dose or prolonged glucocorticoids are associated with hyperglycemia, infection, and delayed wound healing [13,14]. In contrast, low-dose dexamethasone (5 mg) has shown favorable efficacy and safety. The DeLiT trial reported a 30% reduction in postoperative high-sensitivity C-reactive protein (hsCRP) [5]. Within thoracic surgery, the SURTHODEX cohort study revealed that intraoperative dexamethasone administration was associated with a 42% reduction in the risk of postoperative acute respiratory failure and a 24% reduction in the risk of composite infectious complications [8]. Similarly, in colorectal surgery, a single low dose of dexamethasone has been shown not to increase the risk of surgical site infection [15]. Collectively, low-dose dexamethasone may attenuate pe-

rioperative inflammation and reduce respiratory complications while maintaining a good safety profile.

However, the specific association of prophylactic low-dose dexamethasone with PPCs and recovery in TBAD surgery has not been established. Therefore, this retrospective cohort study aims to evaluate the associations between a single 5-mg intravenous dose of dexamethasone (given after anesthesia induction) and PPC incidence, mechanical ventilation duration, ICU and hospital stay, inflammatory markers, and safety outcomes in patients undergoing TBAD surgery.

Methods

Study Design and Population

This single-center, retrospective cohort study was designed to evaluate the association between prophylactic low-dose glucocorticoid administration and postoperative pulmonary complications in patients undergoing surgery for type B aortic dissection. A total of 240 patients who underwent surgical treatment for TBAD (involving the descending aorta, with partial extension to the thoracoabdominal aorta) at our institution between January 2023 and December 2025 were enrolled. Patients were assigned to an intervention group ($n = 118$) or a control group ($n = 122$) based on whether they received low-dose glucocorticoids (5 mg dexamethasone intravenously) before or during surgery. The study was approved by the Ethics Committee of Taizhou Hospital of Zhejiang Province Affiliated to Wenzhou Medical University (approval number: K202603128). All procedures were conducted in accordance with the principles of the Declaration of Helsinki. Given the retrospective nature of the study and the anonymization of all patient data, the requirement for individual informed consent was waived by the Ethics Committee.

Inclusion criteria were: (1) imaging-confirmed diagnosis of type B aortic dissection; (2) age ≥ 18 years; (3) underwent open descending aortic surgery or TEVAR; (4) complete clinical data available with 30-day follow-up.

Exclusion criteria were: (1) severe preexisting pulmonary disease (Chronic Obstructive Pulmonary Disease Global Initiative for Chronic Obstructive Lung Disease (COPD GOLD) stage III–IV, pulmonary fibrosis, etc.); (2) long-term use of glucocorticoids or immunosuppressants; (3) preoperative infection or multi-organ failure; (4) pregnancy or lactation; (5) poorly controlled diabetes (Hemoglobin A1c (HbA1c) $>7.5\%$); (6) active gastric or duodenal ulcer; (7) history of steroid allergy.

Intervention: In the intervention group, dexamethasone 5 mg (1 mL) was administered intravenously as a single bolus over 30 seconds immediately following anesthesia induction. Both groups received the same standard perioperative management, including volume-controlled mechanical ventilation, goal-directed fluid therapy, and multimodal analgesia. Blood glucose was monitored at anesthesia induction, 2 h, 6 h, and 12 h postoperatively; insulin was administered for blood glucose levels >180 mg/dL.

The control group received standard perioperative management without glucocorticoid administration, including volume-controlled mechanical ventilation (tidal volume 6–8 mL/kg predicted body weight, positive end-expiratory pressure (PEEP) 5–8 cmH₂O), goal-directed fluid therapy, and multimodal analgesia (opioids + non-steroidal anti-inflammatory drugs (NSAIDs)). Treatment assignment was not randomized; the decision to administer dexamethasone was made by the attending anesthesiologist based on clinical judgment. To mitigate selection bias, propensity score matching was performed as described below. All patients underwent standardized anesthesia and surgical protocols throughout the perioperative period.

Data Collection

Study data were retrospectively retrieved from the hospital's electronic medical record system. Two researchers, who underwent uniform training and were blinded to group allocation, independently extracted the following information: ① baseline characteristics, including age, sex, body mass index, smoking history, and comorbidities (e.g., hypertension, diabetes); ② surgery-related information, including type of surgery, operative duration, estimated intraoperative blood loss, red blood cell transfusion volume, and use of cardiopulmonary bypass; ③ study endpoint measures. The primary endpoint was the incidence of pulmonary complications within 30 days postoperatively. PPCs were defined according to the European Perioperative Clinical Outcome (EPCO) definitions, encompassing any of the following events: (1) acute respiratory distress syndrome (ARDS, Berlin definition); (2) radiologically confirmed pneumonia requiring treatment; (3) atelectasis requiring clinical intervention (radiologically confirmed atelectasis requiring bronchoscopy, chest physiotherapy, non-invasive positive pressure ventilation, or postural drainage); (4) radiologically or clinically confirmed pulmonary edema. Secondary endpoints included total duration of mechanical ventilation, intensive care unit (ICU) length of stay, total postoperative hospital stay, and inflammatory and oxygenation parameters measured 24 hours postoperatively while patients were receiving mechanical ventilation or standard oxygen therapy. ④ Exploratory safety outcomes, including postoperative blood glucose levels, new-onset infections, and wound healing status. All extracted data were independently cross-verified, and any discrepancies were adjudicated by a third researcher. For PPCs, the two researchers independently assessed each case according to EPCO criteria and were blinded to group allocation. The inter-rater agreement Kappa value was 0.85.

Propensity Score Matching

To minimize selection bias and balance potential confounding variables between the two groups, propensity score matching (PSM) was performed. Propensity scores were estimated using a multivariable logistic regression model

based on baseline characteristics that could influence treatment assignment or outcomes. The following covariates were included: age, sex, body mass index (BMI), diabetes, smoking history, preoperative CRP, preoperative PaO₂/FiO₂ ratio, operative time, blood transfusion volume, and surgical procedure (TEVAR vs. open surgery). Patients in the intervention group were matched 1:1 to those in the control group using nearest neighbor matching without replacement, with a caliper width of 0.2 times the standard deviation of the propensity score. A random seed was used to ensure reproducibility. The matching process was implemented using the MatchIt package in R software (version 4.2.1; R Foundation for Statistical Computing, Vienna, Austria). Standardized mean differences (SMD) were calculated to assess covariate balance before and after matching, with an absolute SMD <0.1 considered indicative of adequate balance. After matching, 93 well-balanced pairs (186 patients) were retained for subsequent analyses.

Statistical Analysis

All statistical analyses were performed using R software (version 4.5.2; R Foundation for Statistical Computing, Vienna, Austria). After PSM, comparisons were performed using statistical methods appropriate for paired data to account for the 1:1 matched design. The normality of continuous variables was assessed using the Shapiro–Wilk test. As all continuous variables showed non-normal distributions, they were presented as median (interquartile range, IQR), and appropriate non-parametric tests were applied for group comparisons. Categorical variables were expressed as numbers (percentages) and compared using the Pearson χ^2 test or Fisher's exact test, as appropriate. A p -value < 0.05 was considered statistically significant.

Results

Baseline Characteristics

Before propensity score matching, a total of 240 patients were enrolled, including 118 patients in the intervention group and 122 patients in the control group. Prior to matching, significant differences were observed between the two groups in age, BMI, preoperative CRP, preoperative PaO₂/FiO₂ ratio, and operative time (all p < 0.05), indicating potential selection bias (Table 1). After 1:1 nearest neighbor matching, 93 well-balanced pairs (186 patients) were successfully obtained. Following matching, the absolute standardized mean differences (SMD) for all covariates were less than 0.1, and no statistically significant differences were observed between groups (all p > 0.05), suggesting good balance of baseline characteristics (Table 1).

Postoperative Pulmonary Complications (PPCs)

After matching, the overall incidence of PPCs was 20.43% in the intervention group, which was significantly lower than 34.41% in the control group (p = 0.033) (Table 2). For specific PPC subtypes, the intervention group showed nu-

Table 1. Baseline characteristics before and after propensity score matching.

Variable	Before matching				After matching			
	Control group (n = 122)	Intervention group (n = 118)	<i>p</i>	SMD	Control group (n = 93)	Intervention group (n = 93)	<i>p</i>	SMD
Age (years), M (Q ₁ , Q ₃)	58.00 (51.00, 64.75)	55.50 (49.50, 61.00)	0.018	0.353	57.00 (50.00, 62.00)	57.00 (52.00, 62.00)	0.503	0.081
BMI (kg/m ²), M (Q ₁ , Q ₃)	25.70 (23.85, 27.37)	24.35 (22.52, 26.20)	<0.001	0.728	25.10 (23.40, 26.70)	24.90 (23.40, 26.80)	0.734	0.058
Preoperative CRP (mg/L), M (Q ₁ , Q ₃)	6.75 (4.62, 8.67)	5.65 (4.10, 7.70)	0.001	0.571	6.10 (4.40, 7.80)	6.10 (4.40, 7.90)	0.913	0.021
Preoperative PaO ₂ /FiO ₂ , M (Q ₁ , Q ₃)	369.50 (354.00, 395.00)	389.00 (363.25, 410.00)	<0.001	0.503	378.00 (358.00, 401.00)	384.00 (358.00, 401.00)	0.530	0.091
Sex, n (%)			0.576	0.032			0.618	0.032
Male	86 (70.49)	87 (73.73)			67 (72.04)	70 (75.27)		
Female	36 (29.51)	31 (26.27)			26 (27.96)	23 (24.73)		
Diabetes, n (%)			0.846	0.010			0.857	0.011
No	97 (79.51)	95 (80.51)			73 (78.49)	74 (79.57)		
Yes	25 (20.49)	23 (19.49)			20 (21.51)	19 (20.43)		
Smoking history, n (%)			0.357	0.059			0.883	0.011
No	61 (50.00)	66 (55.93)			49 (52.69)	50 (53.76)		
Yes	61 (50.00)	52 (44.07)			44 (47.31)	43 (46.24)		
Blood transfusion volume (mL), mean ± SD	425.34 ± 191.32	426.97 ± 199.35	0.948	0.008	414.05 ± 188.66	427.54 ± 196.99	0.634	0.068
Operation time (min), M (Q ₁ , Q ₃)	216.00 (182.25, 249.75)	202.50 (169.50, 234.00)	0.038	0.248	203.00 (177.00, 246.00)	208.00 (172.00, 237.00)	0.809	0.013
TEVAR, n (%)			0.691	0.019			0.848	0.011
No	22 (18.03)	19 (16.10)			16 (17.20)	17 (18.28)		
Yes	100 (81.97)	99 (83.90)			77 (82.80)	76 (81.72)		

Note: BMI, body mass index; CRP, C-reactive protein; TEVAR, thoracic endovascular aortic repair; SMD, standardized mean differences.

Table 2. Postoperative pulmonary complications.

Variables	Control group (n = 93)	Intervention group (n = 93)	p
Overall PPCs, n (%)			0.033
No	61 (65.59)	74 (79.57)	
Yes	32 (34.41)	19 (20.43)	
ARDS, n (%)			0.351
No	86 (92.47)	89 (95.70)	
Yes	7 (7.53)	4 (4.30)	
Pneumonia, n (%)			0.203
No	82 (88.17)	87 (93.55)	
Yes	11 (11.83)	6 (6.45)	
Pulmonary edema, n (%)			0.266
No	84 (90.32)	88 (94.62)	
Yes	9 (9.68)	5 (5.38)	
Other PPCs, n (%)			1.000
No	88 (94.62)	89 (95.70)	
Yes	5 (5.38)	4 (4.30)	

Note: PPCs, postoperative pulmonary complications; ARDS, acute respiratory distress syndrome.

Table 3. Perioperative recovery parameters.

Variables	Control group (n = 93)	Intervention group (n = 93)	p
Duration of mechanical ventilation (h), M (Q ₁ , Q ₃)	18.50 (16.60, 19.70)	17.70 (16.30, 18.90)	0.008
Total hospital stay (d), M (Q ₁ , Q ₃)	15.00 (13.00, 18.00)	15.00 (13.00, 16.00)	0.009
Length of ICU stay (d), M (Q ₁ , Q ₃)	4.00 (3.00, 4.00)	3.00 (3.00, 4.00)	0.003

ICU, intensive care unit.

merically lower incidence rates of ARDS, pneumonia, and pulmonary edema; however, these differences did not reach statistical significance (all $p > 0.05$).

Perioperative Recovery Parameters

Compared with the control group, patients in the intervention group had significantly shorter duration of mechanical ventilation ($p = 0.008$), shorter length of ICU stay ($p = 0.003$), and shorter total hospital stay ($p = 0.009$) (Table 3).

Postoperative Inflammatory and Blood Gas Parameters

At 24 hours postoperatively, the intervention group demonstrated significantly lower CRP levels (median 51.50 mg/L vs. 57.20 mg/L, $p < 0.001$) and white blood cell (WBC) counts (median $10.90 \times 10^9/L$ vs. $12.10 \times 10^9/L$, $p < 0.001$) compared with the control group. Additionally, the PaO₂/FiO₂ ratio was significantly higher in the intervention group (median 310.00 vs. 286.00, $p < 0.001$), indicating attenuated inflammatory response and improved oxygenation (Table 4).

Safety Outcomes

No statistically significant differences were observed between the two groups in terms of postoperative new-onset infection, impaired wound healing, or hyperglycemia (all $p > 0.05$) (Table 5), suggesting that a single low-dose dexamethasone administration was not associated with an increased risk of short-term adverse events.

Discussion

The present study found that perioperative prophylactic administration of a single low dose of dexamethasone was associated with a lower incidence of PPCs following TBAD surgery ($p = 0.033$). Furthermore, it was associated with significant improvements in several key perioperative recovery parameters, including durations of mechanical ventilation, ICU stay, total hospital stay, and postoperative inflammatory and oxygenation levels. This finding aligns with observations from cardiac and thoracic surgery, where low-dose glucocorticoids were associated with suppression of inflammatory responses and accelerated postoperative functional recovery [6,7,8].

Notably, the incidence rates of specific PPCs, such as ARDS and pneumonia, exhibited numerical decreasing trends, supporting the hypothesis that glucocorticoids may mitigate pulmonary inflammatory injury. The significantly reduced postoperative CRP and WBC levels observed in the intervention group support this pharmacological mechanism. While CRP and WBC are commonly used markers of inflammation, WBC count may also be directly influenced by glucocorticoid administration independent of inflammatory response. Therefore, the observed reduction in WBC should be interpreted cautiously. Attenuation of the perioperative inflammatory response is closely linked to a reduced risk of postoperative organ dysfunction. In patients undergoing surgery for aortic dissection, previous reviews have identified activation of inflammatory signal-

Table 4. Postoperative inflammatory and oxygenation parameters at 24 hours.

Variables	Control group (n = 93)	Intervention group (n = 93)	p
CRP (mg/L), M (Q ₁ , Q ₃)	57.20 (51.10, 66.00)	51.50 (44.50, 56.10)	<0.001
WBC ($\times 10^9$ /L), M (Q ₁ , Q ₃)	12.10 (10.40, 13.60)	10.90 (9.10, 12.30)	<0.001
PaO ₂ /FiO ₂ , M (Q ₁ , Q ₃)	286.00 (261.00, 319.00)	310.00 (289.00, 339.00)	<0.001

WBC, white blood cell.

Table 5. Postoperative safety outcomes.

Variables	Control group (n = 93)	Intervention group (n = 93)	p
New-onset infection, n (%)			0.799
No	84 (90.32)	85 (91.40)	
Yes	9 (9.68)	8 (8.60)	
Impaired wound healing, n (%)			1.000
No	88 (94.62)	89 (95.70)	
Yes	5 (5.38)	4 (4.30)	
Hyperglycemia, n (%)			0.650
No	83 (89.25)	81 (87.10)	
Yes	10 (10.75)	12 (12.90)	

ing pathways as a key mechanism underlying postoperative organ dysfunction, including impaired oxygenation, suggesting that effective control of inflammation may mitigate the risk of these complications [16]. In the cardiac surgery domain, multiple reviews have consistently identified systemic inflammatory response as a key contributor to postoperative multi-organ dysfunction [17,18]. This body of evidence provides a theoretical rationale for employing anti-inflammatory strategies to ameliorate postoperative organ function in high-risk patients undergoing major vascular surgery. The improved oxygenation index observed in our study aligns with the established pharmacological mechanisms of glucocorticoids, which involve attenuation of microvascular permeability and suppression of systemic inflammatory responses [5,19,20]. Within the fields of vascular and cardiac surgery, multiple studies have demonstrated that perioperative glucocorticoid administration significantly reduces inflammatory markers such as IL-6 and CRP, while improving postoperative oxygenation and clinical recovery trajectories [7,10,19,20].

Regarding secondary endpoints, the observed reductions in mechanical ventilation duration and hospital length of stay carry substantial clinical significance. This benefit may be attributable to the attenuation of the inflammatory response, which can improve pulmonary compliance and reduce aberrant respiratory drive, thereby facilitating earlier weaning from mechanical ventilation. A shorter duration of mechanical ventilation may reduce the risk of ventilator-associated pneumonia and improve clinical outcomes, as supported by existing literature [21,22,23,24].

However, the results of this study must be interpreted within the context of the following limitations. First, as a retrospective study, this study remains susceptible to residual confounding despite balanced baseline characteristics. The potential influence of unmeasured confounders cannot be completely excluded, including inter-surgeon variability, differences in perioperative fluid management, trans-

fusion triggers, and ventilator settings, all of which may influence PPCs. Second, although PPCs were defined according to standardized EPCO criteria, retrospective ascertainment may introduce diagnostic bias. Moreover, the sample size after PSM (186 patients) may be underpowered to detect differences in rare PPC subtypes such as ARDS, increasing the risk of Type II error, especially given that no formal sample size calculation was performed due to the exploratory nature of this study. Additionally, key inflammatory mediators, such as IL-6 and TNF- α , were not measured, limiting mechanistic validation of the anti-inflammatory effect. Furthermore, only short-term outcomes within 30 days were assessed; long-term outcomes, such as late pulmonary dysfunction, aortic remodeling, and long-term mortality, were not evaluated. Similarly, safety outcomes were limited to short-term events, and delayed adverse outcomes including gastrointestinal bleeding, thromboembolism, or persistent hyperglycemia were not investigated. Another notable limitation is that this is a single-center study with a selected surgical population, which limits the generalizability of the findings to other centers, different surgical teams, or patients with more severe preoperative organ dysfunction. External validation is therefore needed. Finally, although our propensity score model included intraoperative variables, such as operative time and blood transfusion volume, under the premise that dexamethasone administration preceded these events, we acknowledge that the possibility of overadjustment or mediation bias cannot be entirely excluded. Future studies should pre-specify the temporal ordering of covariates in the matching model.

Our study evaluated only a single low-dose (5 mg) dexamethasone regimen. Evidence indicates that the impact of glucocorticoids on the perioperative immune environment differs substantially between single low-dose and multiple or high-dose regimens. A retrospective study by Yang et al. [25], in patients undergoing lung cancer surgery demon-

strated that a single intraoperative 5-mg dexamethasone dose had minimal effects on peripheral blood lymphocyte subsets and function [25]. In contrast, multiple doses or high-dose regimens (15–20 mg dexamethasone equivalent) administered within 24 hours were associated with significant decreases in T-cell and B-cell counts, indicating impaired immune function [25]. This finding suggests that in the TBAD population, a single low-dose regimen may confer a more favorable immunological safety profile while retaining anti-inflammatory efficacy. Future studies should compare different glucocorticoid protocols, including varied timing, dosages, and single versus multiple doses, to determine the optimal regimen for TBAD surgery. Additionally, prospective multicenter randomized controlled trials are needed to confirm these findings and to evaluate hard clinical endpoints such as 30-day mortality and reintubation rates. Biomarker-guided individualized treatment strategies (e.g., based on preoperative inflammatory status) represent a promising direction for precision perioperative medicine.

This study did not identify a significant increase in postoperative infections or impaired wound healing events in the intervention group. This finding is consistent with conclusions regarding the favorable safety profile of single low-dose dexamethasone in major abdominal surgery. A post-hoc analysis by Dahl et al. [26] involving 1386 patients undergoing laparotomy revealed no significant association between perioperative dexamethasone use and surgical site infections or wound-related complications. The PADDI trial further corroborated these findings in 8880 patients undergoing elective major surgery, demonstrating that a single 8-mg dexamethasone dose was non-inferior to placebo regarding the 30-day incidence of surgical site infection [15]. Nevertheless, the risks associated with long-term or high-dose glucocorticoid use warrant continued vigilance [13,14].

Future research directions should include the design of prospective, multicenter RCTs to definitively establish the net benefit of low-dose glucocorticoids in this specific patient population. Additionally, investigations into biomarker-guided individualized treatment strategies are warranted. Such approaches could identify patient subgroups most likely to benefit based on preoperative inflammatory status [27]. This precision-oriented direction has garnered attention within the aortic dissection field. An accompanying editorial to the RCT [9] noted the substantial inter-individual variability in postoperative inflammatory response intensity following aortic dissection surgery. It emphasized that future research should focus on identifying specific patient cohorts most likely to benefit from anti-inflammatory interventions [9]—a paradigm consistent with the evolving principles of precision perioperative medicine.

Conclusions

In conclusion, among patients undergoing surgical repair for type B aortic dissection, perioperative administration of a single low dose of dexamethasone was associated with a lower incidence of PPCs. Furthermore, this intervention was associated with shorter durations of mechanical ventilation and hospitalization, lower postoperative inflammatory marker levels, and improved oxygenation. However, as this was a single-center retrospective study, these findings require confirmation in prospective, multicenter investigations.

Availability of Data and Materials

The data analyzed are available from the corresponding authors upon reasonable request.

Author Contributions

DXZ, XLH, and HW designed the research study. DXZ and YX performed the research. WJZ and CHC analyzed the data. HW drafted the article. All authors have been involved in revising the manuscript critically for important intellectual content. All authors gave final approval of the version to be published. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

The study was approved by the Ethics Committee of Taizhou Hospital of Zhejiang Province Affiliated to Wenzhou Medical University (approval number: K202603128), and all procedures followed the principles of the Declaration of Helsinki. Given the retrospective nature of the study and the anonymization of all patient data, the requirement for individual informed consent was waived by the Ethics Committee.

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Conflict of Interest

The authors declare no conflict of interest.

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