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Risk Factors for Preoperative Hypoxemia in Stanford Type A Aortic Dissection

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Abbreviations: ATAAD: Stanford Type A Acute Aortic Dissection; AD: Aortic Dissection;AAD: Acute Aortic Dissection; ALI: Acute Lung Injury;OSAS: Obstructive Sleep Apnea Syndrome;BMI: Body Mass Index;ERV: Expiratory Reserve Volume (ERV); IL-1β: Interleukin 1 Beta; TNF-α: Tumor Necrosis Factor Alpha; IL-6: Interleukin 6; CRP: C Reactive Protein; WBC: White Blood Cells;Ang-II; Angiotensin II: AT1R; Angiotensin II Receptor Type 1; MMP-9: Matrix Metalloproteinase 9

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1. Abstract

Aortic dissection is a life-threatening cardiovascular condition characterized by the separation of the aortic wall layers due to an intimal tear. It is classified into Stanford Type A and Type B. Type A involving the ascending aorta and carrying a significantly higher risk of mortality and catastrophic complications such as aortic rupture, cardiac tamponade, and end-organ ischemia. Despite advances in surgical and perioperative management, preoperative hypoxemia remains a prevalent and concerning complication in patients with Stanford Type A acute aortic dissection (ATAAD), contributing to poor postoperative outcomes and increased mortality. Preoperative hypoxemia in ATAAD can result from multiple interrelated pathophysiological mechanisms, including cardiopulmonary dysfunction, systemic inflammatory responses, pulmonary congestion, and impaired gas exchange due to malperfusion syndromes. Understanding the risk factors associated with preoperative hypoxemia is critical for optimizing early recognition, risk stratification, and targeted interventions to improve patient outcomes. By synthesizing the latest evidence on this critical perioperative challenge, this review aims to comprehensively examine recent advancements in research regarding the predictors and pathophysiological mechanisms underlying preoperative hypoxemia in ATAAD patients and provide clinicians with an updated framework for assessing and managing preoperative hypoxemia in ATAAD patients, ultimately contributing to improved perioperative care and surgical outcomes.

2. Introduction and Background

Aortic Dissection (AD) refers to the disruption of the elastin-rich media of the aorta due to various etiologies, resulting in weakened resistance of the vessel wall to transverse shear stress of blood flow, which leads to intimal tearing. Blood enters the wall, forming a false lumen that communicates with the true lumen of the aorta through one or more ruptures, creating a dissection [1]. When this occurs within two weeks, it is termed Acute Aortic Dissection (AAD), one of the most severe cardiovascular diseases. After the onset of symptoms, if not treated promptly, the mortality rate increases by 1-2% per hour [2]. AAD can be classified into Stanford type A and Stanford type B based on the location of the primary tear. Stanford type A dissection originates in the ascending aorta, while Stanford type B dissection originates in the descending aorta beyond the left subclavian artery. AAD can be classified into Stanford type A and Stanford type B based on the location of the primary tear. Stanford type A dissection originates in the ascending aorta, while Stanford type B dissection originates in the descending aorta beyond the left subclavian artery [3]. Nearly all diagnosed cases of type A aortic dissection require surgical intervention due to their propensity for life-threatening complications such as aortic rupture, shock, visceral ischemia, cardiac tamponade, and circulatory failure. Patients with type A AAD often suffer from Acute Lung Injury (ALI), which is caused by damage to alveolar epithelial and capillary endothelial cells due to multiple factors, ultimately leading to acute hypoxic respiratory insufficiency [4]. Hypoxemia, defined as a PaO2/FiO2 ratio ≤ 200, is the most typical symptom in patients with ALI. The occurrence of preoperative hypoxemia is not only detrimental to the perioperative management of patients but also a common complication leading to higher incident of postoperative mortality [5]. To enable early assessment and prompt intervention for patients with preoperative hypoxemia, the following is a review of the risk factors for preoperative hypoxemia in AAD.

3. Risk Factors

Recent studies [3-19] have indicated that patient-related factors such as body mass index (BMI), smoking history, pleural effusion, and pericardial effusion are risk factors for preoperative hypoxemia, providing some clinical guidance for patient assessment and opening new directions for related research (Table 1). However, further investigation on the ¬current findings can provide more information on the risk factors and identification of potential high-risk patients before the onset of disease.

3.1. Body Mass Index

Study by Zhang et al. have shown that a BMI ≥ 25 kg/m² is a risk factor for preoperative hypoxemia [6]. Stephen et al. found that as BMI increases in obese patients, oxygen levels start to decrease [7]. An effect that may be related to the overlap of expiratory reserve volume (ERV) reduction caused by the closure of some lung units and atelectasis during normal tidal breathing. However, there is no correlative study on patients with type A AAD. Obesity can lead to changes in inflammatory cytokine levels, which may be related to the occurrence of hypoxemia in patients [8]. Further studies found that the expressions of IL-1β, TNF-α, IL-6, CRP, and WBC in obese AAD patients were increased significantly. Multiple linear regression

Table 1: Patient related risk factors for preoperative hypoxemia. Table shows the important risk factors that are based on the patient history and lifestyle.

Lifestyle and cardiovascular risk factors	Body Mass Index (BMI), Smoking history, Systolic blood pressure
Pre-existing conditions	Pleural Effusion, Pericardial Effusion,
Others	High blood glucose levels

analysis revealed that IL-1, IL-6, and CRP were closely related to BMI, suggesting that the levels of oxidative stress and inflammatory response in obese AAD patients were significantly elevated [9]. It is speculated that this may be related to the long-term high levels of hypoxia in the white adipose tissue of obese individuals, which promotes the release of proinflammatory cytokines (such as TNF-α, IL-6, IL-8, leptin, and IL-1β) and activates proinflammatory signaling pathways to facilitate the release of inflammatory factors [10]. Meanwhile, elevated levels of IL-1β, TNF-α, and IL-6 can lead to pulmonary insufficiency and hypoxemia, further promoting the release of inflammatory factors from adipocytes, creating a vicious cycle that leads to more severe lung injury [11]. Although this study can explain to some extent that ALI in obese patients with aortic dissection may be related to oxidative stress and inflammation, which opens new directions for future research, but it still has some limitations. For example, the number of obese patients diagnosed with ALI in aortic dissection was relatively small in the study. To elucidate the mechanism by which obesity leads to type A AAD, larger sample sizes and longitudinal studies are needed. Nonetheless, it is certain that controlling weight remains an important means of preoperative patient management.

3.2. Smoking History

History of smoking is an important risk factor for patients with type A AAD [12]. In a study involving 505 patients, a strong correlation was found between patients with hypoxemia due to acute type A aortic dissection, that patients with a higher proportion of hypoxemia had a history of smoking [3].This may be due to weakening of ciliary motility because of long-term smoking, causing goblet cell hyperplasia in the bronchi, increasing mucosal secretion, and reduced tracheobronchial clearance, leading to insufficient alveolar ventilation [13]. Recent research suggests that smoking may cause emphysema by damaging proteasome and autophagy activities, which in turn can cause hypoxemia [14].

3.3. Systolic Blood Pressure

Studies done by Guo Z et al. [15]. Have found a correlation between preoperative systolic blood pressure and the occurrence of hypoxemia, with patients in the hypoxemia group having lower systolic blood pressure [3]. This may be because strict blood pressure control is often used to prevent aortic dissection rupture after the onset of AAD. This strict control of blood pressure can lead to reduced blood flow velocity, making it easier for thrombosis formation. Conversely, hypotension can also reduce tissue perfusion and oxygen supply, further amplifying the release of inflammatory factors and subsequently leading to the occurrence of hypoxemia [15].

3.4. Pleural Effusion

Analysis of clinical cases have identified pleural effusion as another risk factor for preoperative hypoxemia in patients with type A AAD [3]. This may be related to the overexpression and massive accumulation of inflammatory cells in the bodies of AAD patients, damaging the endothelial cells in lung capillary, resulting in excessive fluid entering the pleural cavity resulting in pleural effusion [16]. The specific mechanism yet remains to be elucidated. Study found that pleural effusion and pericardial effusion are not independent risk factors for hypoxemia, but the occurrence of pleural effusion may be related to the severity or prolonged time interval from the onset of dissection to treatment [17]. Factors such as atelectasis, pulmonary edema, and reduced tidal volume associated with these conditions may all contribute to hypoxemia. However, it is certain that the presence of a large amount of pleural effusion can lead to decreased lung volume, decreased chest wall compliance, mediastinal shift, and impaired respiratory function, thereby causing hypoxemia.

3.5. Pericardial Effusion

Studies indicate that pericardial effusion is a risk factor for preoperative hypoxemia in patients with type A AAD [3]. Patients with type A AAD accompanied by pericardial effusion often have bilateral or unilateral pleural effusions. This may be caused by an inflammatory exudative reaction surrounding the affected aorta [18]. Along with certain correlation between pericardial effusion and the occurrence of hypoxemia, it is also an independent risk factor for increased mortality in AAD patients [19]. This may be due to the lower systolic blood pressure and pulse pressure in AD patients with pericardial effusion, leading to insufficient lung perfusion and an imbalance in ventilation/perfusion ratio, leading to hypoxemia. A clinical study with 100 patients found that early diagnosis and treatments were crucial to decrease mortality and improve the prognosis in patients with aortic dissection complicated with pericardial effusion [20]. Studies with larger sample sizes are needed further to confirm this.

3.6. Blood Glucose

Blood glucose levels at the time of onset of acute aortic dissection have shown a significant correlation with various cardiovascular outcomes, including hypoxemia. Studies indicate that patients presenting with increased blood glucose levels during an acute aortic dissection event are more prone to the risk of adverse complications, including prolonged mechanical ventilation, hypoxemia and mortality [21]. AD can cause stress-induced hyperglycemia, which may be a manifestation of the inflammatory response under stress conditions and can ultimately result in hypoxemia [22] w. This relationship provides a new direction for clinical research.

4. Disease Risk Factors

The AAD itself and the stress response of the body after onset significantly affect the patient's respiratory function, and AAD patients with certain accompanying diseases and comorbidities are more prone to preoperative hypoxemia. Copious studies [3–5,11,14,15,17–19,22–24,26–41] have explored the related factors (Table 2) and further analyzed the molecular mechanisms of preoperative hypoxemia in AAD patients, providing direction for early risk assessment and intervention measures in clinical patients with preoperative hypoxemia

4.1. Inflammatory Response

Various inflammatory indicators in patients with type A AAD, such as CRP, WBC, IL-6, and MMP-9 levels, are significantly correlated with the occurrence of preoperative hypoxemia [2,3,15,18,19,21–23,27,42]. Patients with acute type A aortic dissection exhibit abnormal manifestations such as aortic dissection, tearing and intimal rupture, with extensive exposure of a large amount of extracellular matrix in the middle layer of the vessel wall and the blood. Neutrophils, monocytes, and macrophages are massively recruited and activated, and the release of related inflammatory factors, such as elastase, reactive oxygen species, etc., causes damage to the lung capillary bed, leading to interstitial lung edema and the extravasation of a large amount of fluid into the alveoli. This increases diffusion thickness and decreases the diffusion membrane area, resulting in hypoxemia and acute lung injury [23]. CRP has been proven to be an independent risk factor for preoperative hypoxemia in patients [24]. CRP is an acute-phase reactant of C-polysaccharide substances synthesized by the liver. When the body is injured or stressed, serum concentration of CRP rapidly increases [25]. CRP stimulates endothelial cells to

Table 2: Disease related risk factors for preoperative hypoxemia. Table includes the risk factors that develop with the disease progression.

Systemic risk factors	Inflammatory response, Disruption of the Coagulation System
Disease Severity	Severity of Aortic dissection, degree of aortic valve insufficiency
Others	Obstructive Sleep Apnea Syndrome (OSAS)

secrete and express adhesion molecules and interleukins, allowing more monocytes to enter the subendothelial space and become macrophages, activating complement and participating in cell apoptosis. It can be used as a sensitive inflammatory marker to indicate the degree of inflammatory response in the body [25]. CRP ≥ 11.21 mg/L has been proven to be an important risk factor for preoperative hypoxemia and can be used as a predictor of in-hospital mortality risk in ATAAD patients [26]. Studies have also found that preoperative administration of 'ulinastatin' in patients with type A AAD to inhibit the inflammatory response does not result in change in perioperative conditions but significantly improves postoperative hypoxemia, providing new focus for research on medication and treatment [24]. After the occurrence of aortic dissection, the patient's oxygenation index gradually decreases, while serum IL-6 levels rapidly rise to peak levels [24, 27]. Subsequently, as the patient's inflammatory response weakens, the oxygenation index shows an upward trend. The level of serum IL-6 can predict the prognosis of patients [24]. As a highly effective proinflammatory factor, IL-6 plays an important role in regulating the inflammatory response. At the same time, a continuous rise in IL-6 can easily trigger acute respiratory distress syndrome, leading to hypoxemia [27]. The incidence of hypoxemia is significantly increased in patients with an increased WBC count [28]. Detecting arterial blood WBC count is a simple and quick means of clinically assessing the inflammatory status in patients, reflecting the degree of inflammatory response in the blood to a certain extent. Many WBCs can accumulate in the lungs, leading to inflammatory response, plural effusion and alveolar edema, which affect the lung's gas exchange function and ultimately result in the manifestation of hypoxemia. Current studies [26, 29], have shown that serum MMP-9 levels are significantly elevated in patients with AAD complicated with ALI, and the expression of MMP-9 in lung tissue originates from macrophage. In animal models, the release of MMP-9 by macrophages ultimately leads to ALI, a process that may be related to Ang-II mediating the release of MMP-9 in lung tissue macrophages [30]. Early application of AT1R blockers and MMP inhibitors can prevent or alleviate the onset and progression of ALI [31]. However, there are still certain limitations in this research. Animal models cannot fully simulate the pathogenesis of AAD complicated with ALI. To investigate whether MMP-9 is a risk factor for preoperative hypoxemia in patients with type A AAD, further clinical correlation trials with human patients are needed, giving new research opportunities.Therefore, preoperative anti-inflammatory treatment for patients with type A AAD may improve their hypoxemia, thereby enhancing surgical tolerance and improving patient prognosis.

4.2. Disruption of the Coagulation System

Multiple studies have found that D-dimer levels are elevated in the hypoxemia group, suggesting greater platelet consumption compared to the non-hypoxemia group [32]. D-dimer is a product released during the degradation of cross-linked fibrin polymer by plasmin and is involved in both coagulation and fibrinolysis processes. It serves as an ideal molecular marker for reflecting coagulation function and fibrinolysis hyperfunction [33]. Multiple studies [26, 32, 34], have shown that the onset of AD is often accompanied by abnormalities in coagulation function, with activation of the fibrinolytic system and the formation of microthrombi in the body, leading to increased D-dimer levels. Factors such as damage to the aortic intima, exposure of subendothelial tissue, blood flow through the endothelium-free false lumen, and instability of the vascular wall can all induce the activation of procoagulant substances. Abnormal blood flow changes caused by the formation of the false lumen can produce mechanical damage to platelets, resulting in excessive consumption of platelets, fibrinogen, and coagulation factors [34]. This may also be related to the severity of the dissection and the size of the false lumen. Patients with acute aortic dissection have hypercoagulable blood during the perioperative period, which is prone to the formation of microthrombi [34, 35]. When these thrombi embolize the alveolar capillaries, hypoxemia will inevitably occur. Therefore, preoperative anticoagulant therapy for patients may prevent or improve preoperative hypoxemia, thereby improving patient prognosis.

4.3. Severity of Aortic Dissection

The larger the false lumen of the dissection and the greater its proportion of aortic volume, the smaller the PaO2/FiO2 ratio [36]. When the dissection is bigger in size it can cause low perfusion in the pulmonary circulation caused by blood entering the false lumen. This can lead to hypoperfusion of systemic arteries including the superior mesenteric artery and bilateral renal arteries, increasing the risk of hypoxemia. The area of false lumen is an independent predictor of hypoxemia and is an independent risk factor for prognosis [36]. This is closely related to peak levels of serum CRP and white blood cell counts in the blood, indicating that the inflammatory response plays an important role in this process.

4.4. Obstructive Sleep Apnea Syndrome (OSAS)

Obstructive sleep apnea syndrome (OSAS) is a common but underestimated potentially dangerous disease. It is characterized by repeated complete or partial collapse of the upper airway during sleep, leading to airflow interruption and increased inspiratory resistance [37]. Despite increased respiratory effort, obstructive hypoventilation or apnea caused by the collapse of the upper airway ultimately affects sleep structure and overall health status through both immediate and long-term mechanisms. OSAS can result in decreased oxygen partial pressure and carbon dioxide retention, leading to intermittent hypoxemia [38]. Long-term intermittent hypoxemia may cause ventilation/perfusion mismatch in patients, inducing hypoxemia. Recent research by Liu et al. [39] has found that intermittent hypoxemia caused by OSAS, can exacerbate the condition of AAD patients through the ROS-HIF-1α-MMPs mechanism. Additionally, it was discovered that the HIF-1α inhibitor KC7F2 improved the toxic effects of intermittent hypoxemia on AD mice, providing a new therapeutic approach for clinical patients with AAD complicated by OSAS.

4.5. The Degree of Aortic Valve Insufficiency

Research [24] has indicated that the varying degree of aortic valve insufficiency exhibits differing levels of risk for developing preoperative hypoxemia in patients with Type A AAD. Aortic valve insufficiency is often accompanied by the regurgitation of aortic blood, which leads to increased left ventricular end-diastolic pressure and filling pressure, exceeding left atrial pressure and resulting in premature closure of the mitral valve during diastole. This volume overload reduces the efficiency of the heart to pump oxygenated blood effectively and results in diminished levels of oxygen in the bloodstream [40]. As left ventricular decompensation progresses, increased left ventricular diastolic pressure leads to diastolic mitral regurgitation and subsequently elevated pulmonary venous pressure, causing pulmonary edema [41]. Furthermore, due to the limited ability of the ventricle to increase cardiac output, the left ventricle cannot rapidly adapt to increased ventricular volume, ultimately resulting in decreased cardiac output and even myocardial ischemia.For patients with Type A AAD and concurrent aortic valve insufficiency, continuous monitoring of the degree of aortic valve regurgitation is not only of great significance for predicting preoperative hypoxemia but also valuable for assessing postoperative cardiac function. This underscores the importance of comprehensive preoperative assessment and management strategies focused for such patients to optimize their surgical outcomes and postoperative recovery.

5. Conclusion

Preoperative hypoxemia in patients with Stanford Type A acute aortic dissection poses substantial challenges to perioperative management, markedly elevates surgical risks, and adversely impacts postoperative outcomes. This complication and research direction has gained growing recognition and interest on both national and

international levels. Research has explored the pathogenic mechanisms of established risk factors while also investigating emerging factors through exploratory and speculative studies.Nonetheless, many unknowns remain in our complete understanding of the risk factors and optimal interventions for preoperative hypoxemia in patients with Type A Acute Aortic Dissection, warranting further research studies and investigation.

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