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Contents

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OPINION REVIEW

- 3114 Modernising autism spectrum disorder model engineering and treatment *via* CRISPR-Cas9: A gene reprogramming approach

Sandhu A, Kumar A, Rawat K, Gautam V, Sharma A, Saha L

REVIEW

- 3128 Burden of disability in type 2 diabetes mellitus and the moderating effects of physical activity

Oyewole OO, Ale AO, Ogunlana MO, Gurayah T

MINIREVIEWS

- 3140 Postoperative hypoxemia for patients undergoing Stanford type A aortic dissection

Liu HY, Zhang SP, Zhang CX, Gao QY, Liu YY, Ge SL

ORIGINAL ARTICLE

Case Control Study

- 3148 Impact of extended nursing model after multi-disciplinary treatment on young patient with post-stroke

Xu XY, Pang ZJ, Li MH, Wang K, Song J, Cao Y, Fang M

- 3158 Changes and significance of serum ubiquitin carboxyl-terminal hydrolase L1 and glial fibrillary acidic protein in patients with glioma

Zhu QH, Wu JK, Hou GL

Retrospective Study

- 3167 Multitrack and multianchor point screw technique combined with the Wiltse approach for lesion debridement for lumbar tuberculosis

Yuan YF, Ren ZX, Zhang C, Li GJ, Liu BZ, Li XD, Miao J, Li JF

- 3176 Clinical features and prognostic factors in 49 patients with follicular lymphoma at a single center: A retrospective analysis

Wu H, Sun HC, Ouyang GF

- 3187 Value of optical coherence tomography measurement of macular thickness and optic disc parameters for glaucoma screening in patients with high myopia

Mu H, Li RS, Yin Z, Feng ZL

Observational Study

- 3195 Comparative study of the clinical efficacy of all-inside and traditional techniques in anterior cruciate ligament reconstruction

An BJ, Wang YT, Zhao Z, Wang MX, Xing GY

- 3204** Positioning and design by computed tomography imaging in neuroendoscopic surgery of patients with chronic subdural hematoma

Wang XJ, Yin YH, Zhang LY, Wang ZF, Sun C, Cui ZM

- 3211** Evaluation of chronic idiopathic tinnitus and its psychosocial triggers

Hamed SA, Attiah FA, Fawzy M, Azzam M

- 3224** Intestinal complications in patients with Crohn's disease in the Brazilian public healthcare system between 2011 and 2020

Sasaki LY, Martins AL, Galhardi-Gasparini R, Saad-Hossne R, Ritter AMV, Barreto TB, Marcolino T, Balula B, Yang-Santos C

Randomized Controlled Trial

- 3238** Effect of non-pharmacological treatment on the full recovery of social functioning in patients with attention deficit hyperactivity disorder

Lv YB, Cheng W, Wang MH, Wang XM, Hu YL, Lv LQ

CASE REPORT

- 3248** Diagnosis of tuberculous uveitis by the macrogenome of intraocular fluid: A case report and review of the literature

Zhang YK, Guan Y, Zhao J, Wang LF

- 3256** Intragastric fish bones migrate into the liver: A case report

Dai MG, Zheng JJ, Yang J, Ye B

- 3261** Primary seminal vesicle adenocarcinoma with a history of seminal vesicle cyst: A case report and review of literature

Yao Y, Liu S, He YL, Luo L, Zhang GM

- 3267** Immune checkpoint inhibitor therapy-induced autoimmune polyendocrine syndrome type II and Crohn's disease: A case report

Gao MJ, Xu Y, Wang WB

- 3275** Late-onset mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes syndrome with mitochondrial DNA 3243A>G mutation masquerading as autoimmune encephalitis: A case report

Wang JW, Yuan XB, Chen HF

- 3282** Metastatic gastric cancer from breast carcinoma presenting with paraneoplastic rheumatic syndrome: A case report

Rech MB, da-Cruz ER, Salgado K, Balbinot RA, Balbinot SS, Soldera J

- 3288** Novel mutation of SPG4 gene in a Chinese family with hereditary spastic paraplegia: A case report

Wang J, Bu WT, Zhu MJ, Tang JY, Liu XM

- 3295** Chronic pulmonary mucormycosis caused by rhizopus microsporus mimics lung carcinoma in an immunocompetent adult: A case report

Guo XZ, Gong LH, Wang WX, Yang DS, Zhang BH, Zhou ZT, Yu XH

- 3304** Idiopathic sclerosing mesenteritis presenting with small bowel volvulus in a patient with antiphospholipid syndrome: A case report
Chennavasin P, Gururatsakul M
- 3311** *Neisseria mucosa* - A rare cause of peritoneal dialysis-related peritonitis: A case report
Ren JM, Zhang XY, Liu SY
- 3317** Rectal prolapse in a 30-year-old bladder stone male patient: A case report
Ding HX, Huang JG, Feng C, Tai SC
- 3323** Successful treatment of veno-arterial extracorporeal membrane oxygenation complicated with left ventricular thrombus by intravenous thrombolysis: A case report
Wang YD, Lin JF, Huang XY, Han XD
- 3330** Successful remimazolam sedation-epidural block in an older patient with severe chronic obstructive pulmonary disease: A case report
Yu JJ, Pei HS, Meng Y
- 3340** *De novo* mutation of NAXE (APOAIBP)-related early-onset progressive encephalopathy with brain edema and/or leukoencephalopathy-1: A case report
Ding L, Huang TT, Ying GH, Wang SY, Xu HF, Qian H, Rahman F, Lu XP, Guo H, Zheng G, Zhang G
- 3351** Iatrogenic atlantoaxial rotatory subluxation after thyroidectomy in a pediatric patient: A case report
Hong WJ, Lee JK, Hong JH, Han MS, Lee SS
- 3356** Bladder metastasis from epidermal growth factor receptor mutant lung cancer: A case report
Jin CB, Yang L
- 3362** Primary rectal mucosa-associated lymphoid tissue lymphoma treated with only endoscopic submucosal dissection: A case report
Lee WS, Noh MG, Joo YE

ABOUT COVER

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Postoperative hypoxemia for patients undergoing Stanford type A aortic dissection

Hai-Yuan Liu, Shuai-Peng Zhang, Cheng-Xin Zhang, Qing-Yun Gao, Yu-Yong Liu, Sheng-Lin Ge

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Abstract

Clinically, it is widely recognized that surgical treatment is the preferred and reliable option for Stanford type A aortic dissection. Stanford type A aortic dissection is an emergent and serious cardiovascular disease characterized with an acute onset, poor prognosis, and high mortality. However, the incidences of postoperative complications are relatively higher due to the complexity of the disease and its intricate procedure. It has been considered that hypoxemia, one of the most common postoperative complications, plays an important role in having a worse clinical prognosis. Therefore, the effective intervention of postoperative hypoxemia is significant for the improved prognosis of patients with Stanford type A aortic dissection.

Key Words: Stanford type A aortic dissection; Hypoxemia; Risk factors; Intervention; Mortality

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Core Tip: Surgically, it has been considered that in patients suffering with Stanford type A dissection, postoperative complications are the main risk factors that lead to higher mortality and worse outcome. As one of the most common postoperative complications, the importance of prevention and intervention on the postoperative hypoxemia should be fully emphasized with the aim to decrease the mortality and improve the outcome.

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INTRODUCTION

Stanford type A aortic dissection is described as the unstable and fatal rupture of aortic wall involving the ascending aorta and arch. The physio-pathological alteration of Stanford type A aortic dissection is mainly summarized as the separation from media layers leading to false lumen within the wall under the influence of two critical factors, weakness and increased tension of the aortic wall[1]. Once confirmed, among different clinical protocols, the surgical strategy should be predominated and under the guide of strict assessment, the long-term clinical outcome of patients will be improved positively[2, 3]. On the other hand, limited by huge difficulty and complexity of surgical procedure as well as various individual uncertainty of patients, the incidences of postoperative complications are relatively higher. Additionally, hypoxemia caused by multifactors is closely associated with longer mechanical ventilation and hospital stay, progressive respiratory failure, and increased mortality[4].

Therefore, the aim of this study is to provide clinical recommendations for the improvement of postoperative hypoxemia of patients with Stanford type A aortic dissection based on risk factors and intervention of hypoxemia.

RISK FACTORS OF POSTOPERATIVE HYPOXEMIA

Organic disorder

Postoperative stable organic function is positively associated with the inhibition of an inflammatory response. Hence, under the consequence of organic disorder, both internal environment disturbance and inflammatory response are activated. In a retrospective study aiming to explore the independent predictors of hypoxemia, it was found that renal disorder is an independent factor associated with hypoxemia. Activated inflammatory response, unregulated production of erythropoietin, and abnormal delivery of oxygen are also considered possible mechanisms. However, there is no significant correlation between renal insufficiency and mild hypoxemia[5]. Moreover, according to Guo *et al*[6], it has been demonstrated that complicated hypertension is found in the majority of patients with Stanford type A aortic dissection, which leads to atherosclerosis, is negatively associated with pulmonary circulation, and may further induce postoperative hypoxemia. In addition, impairment of the respiratory system by the cascade reactions, originating from aortic dissection, is thought to be pro-inflammatory, which increases the permeability of both endothelial and epithelial cells. Pulmonary vascular pressure affects the physiological function of alveolar surfactant and eventually disturbs normal oxygenation processes[7]. Meanwhile, it has been indicated that angiotensin II is related to the apoptosis of pulmonary microvascular endothelial cells and the upregulated expression of monocyte chemoattractant protein-1 (MCP-1). Angiotensin II is widely considered the key factor during the development of dissection. It exaggerates elastin fragmentation and damages the structure of adventitial layer through activation of caspase-3 and imbalance of the B-cell lymphoma-2 (Bcl-2)/Bcl-2-associated X protein ratio. As a result, both angiotensin II and MCP-1 are critical factors for the inactivated alveolus-capillary barrier and increased pulmonary vascular permeability[8,9].

Obesity

Clinically, during the postoperative stage, the respiratory function of obese patients with Stanford type A aortic dissection is less stable and insufficient to maintain the physiological demands due to limited rehabilitation processes. The lung compliance of obese patients is generally weaker, which means that both breathing difficulties and respiratory resistance are more frequent among the obese population. Obesity is also a driving factor for oxidative stress and reactive oxygen products causing direct deterioration of lung function even hypoxemia[10]. In a retrospective study conducted by Sheng *et al*[11], it has been found that 25 kg/m² of body mass index (BMI) is the threshold value; if exceeded, the assessment of lung function becomes worse and the possibility of complications with postoperative hypoxemia is greater. In another study focusing on the Japanese population, the obese threshold of BMI was set to 30 kg/m², while 25.5-29.0 kg/m² was defined as overweight. It was found that, compared with patients with normal weight, the incidences of ventilation > 48 h for the overweight and obese patients were 60.1% and 78.3%, respectively[12]. Shi *et al*[13] indicated that elevated free fatty acid in obese patients is associated with early postoperative lung damage through the process of endothelial activation and is inversely related to the lowest oxygen level 24 h after cardiac surgery. Furthermore, the level of malondialdehyde is significantly increased in obese patients with aortic dissection, which

indicates that the balance between oxidation and antioxidation is destroyed and sequentially larger secretions and synthesis of inflammatory factors are activated[14]. Meanwhile, patients with obesity are at high risk for obstructive sleep apnea (OSA), and of note, OSA is the risk factor of the development for aortic dissection and postoperative hypoxemia. First, based on findings from a study conducted by Wang *et al*[15], aortic root diameter is positively correlated with the severity of OSA. Consequently, due to the negative effects of adverse apnea events causing failure of the oxidative system, the cascade of inflammatory reactions and release of cytokines such as interleukin (IL)-2, IL-4, and IL-6 are apparent among patients with OSA[16]. Similarly, in a retrospective study focusing on the Chinese patients, the predictive value of OSA on hypoxemia after Stanford type A aortic dissection should be emphasized although the exact mechanism remains unknown[17].

Transfusion of blood products

Since the surgical treatment of Stanford type A aortic dissection is time consuming, considerable blood lost is inevitable during the procedure. Transfusion is necessary to maintain the relative normal level of hemoglobin. However, on the other hand, transfusion-related adverse events including allergy, immune response, infection, circulatory overload, and even organ injury should not be ignored and evaluated with caution. Currently, transfusion-related acute lung injury (TRALI) is the most frequent adverse event that possibly may induce secondary severe postoperative hypoxemia. In a large cohort study enrolling a total of 8944 patients, with the aim of decreasing the incidence of TRALI, it was demonstrated that the mortality of TRALI in the intensive care unit (ICU) was 70%, which is significantly higher than that in other medical units. Hence, it is especially challenging to patients undergoing surgery for Stanford type A aortic dissection, and some improvements have also been observed through the use of solvent/detergent plasma; nevertheless, there are no significant clinical differences[18]. Recently, enhanced coagulation and anticoagulation, as well as damaged fibrinolysis, have been found in the TRALI animal model, through which sequential increased fibrin accumulation in lungs led to platelet capture, the potential risk factor of hypercoagulable state and formation of pulmonary thrombin [19]. Neutrophil extracellular trap (NET), to some extent, is thought to be related to the clinical advancement of TRALI through the impairment of both lung tissue and endothelial cells. Le *et al*[20] found that the inhibition of protective factor, Krüppel-like Factor 2, is induced by microRNA 144 (miR-144) and further activates the nuclear factor-kappa B (NF-κB)/C-X-C motif chemokine receptor 1 signaling pathway, which are possibly responsible for the generation of NET. As another newer mechanism concentrating on the soluble antigen by Bayat *et al*[21], the binding between soluble cluster of differentiation 177/proteinase 3 and platelet endothelial cell adhesion molecule-1 activates endothelial cells and even overrides respiratory functional barrier. In addition to TRALI, transfusion-associated circulatory overload is another potential risk factor to pulmonary edema with clinical presentations including dyspnea, jugular vein engorgement, and elevated systolic blood pressure due to increased pulmonary vascular permeability[22].

Cardiopulmonary bypass

Cardiopulmonary bypass (CPB), an indispensable assisted platform during the surgical procedure of Stanford type A aortic dissection, is critically responsible for the maintenance and protection of normal cardiopulmonary physiological function. Nevertheless, CPB-related adverse events, which are gradually presented following the duration of surgery, include the immune response originating from extracorporeal blood circuit and injury of blood components by compression of the mechanical pump. Lung injury by CPB is mainly concluded as two significant points: ischemia-reperfusion injury and systematic inflammatory reaction. There is a synergistic effect for both, of which, vessels, tissue, and parenchyma of pulmonary are seriously affected. Therefore, a series of pathological alterations of the lungs, predicting postoperative hypoxemia and CPB duration association are observed such as impaired permeability, interstitial edema, fluid accumulation, reduced surface-active substances, and gas exchange disorder [23]. In an observatory study of a rat model, established by Deng *et al*[24], it was shown that high-mobility cassette-1/Toll-like receptor 4 (TLR4)/NF-κB is a reliable signaling pathway that induces CPB-related lung injury and activates a pro-inflammatory effect. Moreover, recently, the association between ferroptosis and CPB-related lung injury was first demonstrated based on the finding that the level of labeled indicators for ferroptosis, glutathione peroxidase 4, and acyl-coenzyme A synthetase long-chain family member 4 is regulated significantly within lung tissue after CPB[25]. As is well known, deep hypothermic circulatory arrest (DHCA) is a special and important period during the entire surgical procedure of Stanford type A aortic dissection, within which a relative bloodless field is provided to increase the possibility of a successful surgery. On the other hand, DHCA is also a risk factor for some postoperative complications including hypoxemia due to ischemia and hypoxia. Kong *et al*[26] suggested that activated autophagy by DHCA is associated with lung injury and its presentation with time-dependent dynamic change characterized as activity decreasing at 3 h after surgery while increasing at 6 h after surgery. Metabolically, especially within the dorsocaudal lung region, CPB/DHCA also affects the regulation pathway of metabolites including amino acids, carbohydrates, lipids, steroids, and vitamins[27].

Other factors

Severe pain and older age, to some extent, are closely correlated with postoperative hypoxemia, whereas reduced muscular strength with a grip < 15 s, due to inadequate evaluation of muscle reversal, is a higher significant predictor contributing to the hypoxemia[28]. Besides, smoking history, chronic respiratory disease, lower oxygenation with arterial oxygen saturation < 96% should be emphasized during the comprehensive assessment for postoperative hypoxemia, and interestingly, different from the obesity mentioned above, the possibility of postoperative hypoxemia for patients with relatively lower BMI < 18.5 kg/m² is also higher than that for those with normal BMI, possibly suggesting that malnourishment and/or other complicated chronic diseases are equally significant for the pathogenesis [29]. In an animal model of acute lung injury, Wu *et al*[30] indicated that the pulmonary edema, alveolar protein leaking, and inflammatory response were found in rats treated with hyperglycemia, which was associated with the upregulated serum-glucocorticoid kinase 1-NKCC1 pathway inhibiting excessive fluid removal and activating the inflammatory response. Morey *et al*[31] demonstrated that the synergy effect between hyperglycemia and hypoxemia is significant for persistent inflammation state delaying or even worsening the clinical outcome.

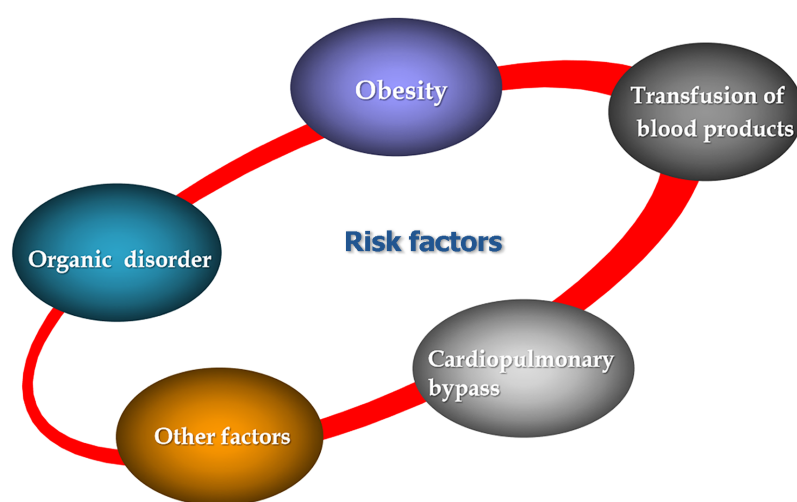
THE INTERVENTION OF POSTOPERATIVE HYPOXEMIA

Medicine intervention

To date, conventionally, medicine intervention should be necessary and compulsory when postoperative hypoxemia occurs. It should predominantly include ulinastatin and sevoflurane, which mainly suppress inflammatory response and improve lung injury. Clinically, ulinastatin, as a regular trypsin inhibitor, is extracted from human urine and used in the treatment of acute or critical inflammatory response and organ functional failure. In accordance with the result of study conducted by Jiang *et al*[32], it has been indicated that the improvement of pulmonary edema by ulinastatin is presented through reduced permeability and enhanced alveolar fluid clearance, whose mechanism may involve two pathways, activated phosphoinositide 3-kinase/Akt and suppressed TLR4/ myeloid differentiation primary response 88/NF- κ B. Meanwhile, enhanced autophagy is also another medical target for relief of lung injury by ulinastatin *via* the upregulation of transforming growth factor- β 1 and light chain 3 and the downregulation of α -smooth muscle actin, matrix metalloproteinase (MMP)-2 and MMP-9[33]. Sevoflurane is used as a classic agent against acute lung injury through multiple pathways. Notably, the suppression of oxidative stress by sevoflurane is firstly elaborated in the mice model of acute lung injury, which is dependent on the pathway of Kelch-like ECH-associated protein 1/nuclear factor erythroid 2-related factor 2[34]. In a similar study, the LINC00839/miR-223/NLR family pyrin domain containing 3 axis was newly confirmed as the driven regulator involving the development of acute lung injury, through which sevoflurane can also be responsible for lung protection[35]. Nitric oxide (NO) has been currently adapted widely for the treatment of respiratory system failure and critical illness such as acute respiratory distress syndrome (ARDS), pulmonary hypertension, and lung transplantation. In a retrospective study focusing on postoperative hypoxemia, the incidence of postoperative hypoxemia for patients who underwent low-dose NO therapy (5-10 ppm) after surgery for Stanford type A aortic dissection decreased and the length of ICU stay as well as duration of mechanical ventilation significantly improved. Of note, the only concern about this unique therapy was prolonged bleeding time due to restrained platelet aggregation and adhesion with endothelium by NO[36]. Furthermore, it has been suggested that NO therapy is especially adaptive to the improvement of postoperative refractory hypoxemia within 72 h after surgery[37,38]. Moreover, some promising results from other attempts have also been evaluated and verified. Prophylactic usage of erythropoietin is valuable in the prevention of lung injury with the exact mechanism, for example, decreasing the levels of negative inflammatory factors such as IL-1 β , tumor necrosis factor- α and NF- κ B; enhancing lung compliance, optimizing gas exchange; and reducing airway pressure[39]. On the other hand, the highly selective and potent α 2 adrenergic agonist, dexmedetomidine, is an effective agent against oxidative stress injury with the aim of protecting lung tissue and maintaining normal physiological function by preserving mitochondrial dynamic equilibrium *via* the hypoxia inducible factor-1 α /heme oxygenase 1 signaling pathway[40]. However, different opinions on the lung protection of dexmedetomidine from Kim *et al* [41] have shown that, regardless of the advantage of anti-oxidative stress presented with the decreasing of malondialdehyde, the increase in urine output and less usage of vasoactive agents, for dexmedetomidine, it does not play an important role on lung protection in surgery.

Supportive intervention

Mechanical ventilation is a necessary support after surgery for Stanford type A aortic dissection. It maintains the stability of vital signs and promotes rehabilitation. Traditionally, supine position is widely accepted as the standard mode of mechanical ventilation, while recently, the prone position is considered the better alternative option. In a meta-analysis conducted by Cao *et al*[42], it has been indicated that, compared with traditional supine position, the mortality of patients undergoing prone position ventilation is lower, especially for the population < 60 years despite the findings of some



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Figure 1 Risk factors of postoperative hypoxemia for patients with Stanford type A aortic dissection.

insignificant adverse events such as pressure ulcer, displacement of thoracotomy tube, and endotracheal tube obstruction. Based on the findings from another comparison study, under the support of prone position ventilation, compared with the supine position, the resting lung volume measured by functional residual capacity and end-expiratory lung volume increased while dynamic strain decreased, and all differences were significant[43]. As concluded, the lung protective and improved oxygenation mechanism of prone position ventilation is explained by the fact that first, lung volume is free from compression by heart; second, the improvement of ventilation/blood ratio, pulmonary shunts are eliminated; and third, the redistribution of edema fluid is influenced by gravity when the pressure changes gradually[44]. Furthermore, Fioretto *et al*[45] optimized the prone position ventilation strategy with high-frequency oscillatory and demonstrated that compared with conventional mechanical ventilation, the optimized strategy was more feasible and reliable in reducing oxidative damages and preventing lung injury. Also, positive end-expiratory pressure (PEEP) is an important mode for the improvement of hypoxemia. It has been suggested that PEEP has the potential to stabilize dependent lung regions at the end-expiration and inhibit inflammatory response during the stage of mechanical ventilation free from the influence of spontaneous breathing[46]. Wu *et al*[47] established a porcine model of ARDS exploring the practical feasibility of transpulmonary pressure guided PEEP. In accordance with this result, under transpulmonary pressure of 25 cm water, the positive effects of PEEP could be observed including compliance improvement, dead space ventilation reduction, and lung protection. In a clinical study for patients with moderate to severe ARDS, when PEEP was combined with prone position, relative lower titration of PEEP was more adaptive and recommended due to increased transpulmonary pressure caused by prone position[48]. However, obese patients with ARDS should be treated reversely and equipped with higher PEEP strategy to improve 60-d all-cause in-hospital mortality[49]. On the other hand, higher PEEP may involve the redistribution balance for both ventilation and perfusion within different lung units to avoid the excessive ineffective ventilation or perfusion, which adjusts the physiological ratio of ventilation/perfusion to optimize regional tidal volume and decrease the risk of lung injury[50]. Necessarily and possibly, even if mechanical ventilation is weaned, hypoxemic respiratory failure is also life threatening due to severe infection, edema, sepsis or ARDS; therefore, alternative high-flow nasal cannula is thought to be responsible for the persistent respiratory function improvement with comfortable acceptance, better airway clearance, and less abdominal distention[51].

CONCLUSION

Hypoxemia is one of the major complications after surgery for Stanford type A aortic dissection. Comprehensively, the pathogenesis and development of postoperative hypoxemia involve the interaction of many risk factors including organic disorder, obesity, transfusion, and CPB (Figure 1). For treatment, the combination between medicine and supportive intervention is considered the more sustainable model. Surgically, all perioperative points should be managed with caution and patience, starting with reasonable preoperative lung function assessment, experienced cooperation of surgical team, and flawless postoperative rehabilitation. The vision, therefore, seeks to have larger scale studies concentrating on the long-term outcome of postoperative hypoxemia and more effective and optimal management.

FOOTNOTES

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