

Unilateral pulmonary edema following minimally invasive cardiac surgery: keeping both eyes *maximally* open



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Over the last decades, advances in minimally invasive cardiac surgery (MICS) have opened up newer avenues for achieving an enhanced recovery by optimizing the cost-benefit ratio in the cardiac surgical arena. This paradigm shift resonates well with the overall aim of an improved quality of care, patient satisfaction and reduction in post-operative stay, favorably modulating the perioperative morbidity [1–3]. While the advantages generally outweigh the risks involved, MICS does however present challenges previously unheard of in similar conventional surgical cohorts [1, 4]. These include: challenged surgical field visualization, requirement of alternate cannulation sites for cardio pulmonary bypass (CPB) and one lung ventilation (OLV). This can potentially translate as increased concerns surrounding perioperative neurological and respiratory complications such as stroke and unilateral pulmonary edema (UPE), respectively [1, 4, 5].

UPE appears to be an often overlooked yet life-threatening complication, specifically associated with MICS. The incidence of UPE in patients undergoing MICS widely ranges from 1.2% to 25% and the associated mortality rate can be as high as 33% depending on type of surgery and ventilation strategies employed [4]. The lack of uniform definition and occult presentation may account for the varied occurrence of UPE reported across studies [4]. However, the most commonly used definition is: a new onset unilateral pulmonary edema (increased right- versus left-sided hemithorax opacification which amounts to > 20% of the chest field, after excluding atelectasis) evident on chest radiograph (CXR) within 24 h after surgery [1, 5]. A study by Kesävuori *et al.* classified UPE in patients with > 25% opacity of hemithorax on CXR into two groups, namely mild and severe, with corresponding signs of interstitial edema and alveolar edema [4]. More importantly, the pathophysiological basis of UPE to date remains unclear, with various suggested mechanisms. These include direct insults from atelectasis and hydrostatic forces leading to an alveolar-capillary barrier disruption, compounded by an indirect injury following ischemia-reperfusion, mediated by oxidative free radicals and chemo-toxic inflammatory cells [1, 4, 5].

Nevertheless, a range of preoperative risk factors for UPE have received attention: advanced age, diabetes mellitus, chronic obstructive pulmonary disease, elevated leucocyte count, serum creatinine and C-reactive protein, etc. [4, 6]. Echocardiographic findings such as pulmonary hypertension, left ventricular ejection fraction < 50%, significant right ventricular dysfunction and a pathological right pulmonary vein Doppler profile are additionally associated with UPE [1, 4–6]. A study by Khalil *et al.* also identified that patients receiving calcium channel blockers, diuretics and corticosteroids were also at an accentuated risk of UPE [6]. Specific to the intraoperative period, fresh frozen plasma transfusion, OLV, invasive mitral valve surgery, especially a robotic approach, use of catecholamines, long surgical duration and CPB or cross-clamp times have been implicated with an increased incidence of UPE [4–6].

Coming to the diagnosis, UPE is primarily recognized on a routine postoperative CXR employing the above-mentioned criterion, given that the former may transpire without initial clinical manifestations [1]. Apart from the radiological evidence, postoperative arterial blood gas analysis demonstrates progressive hypoxia in patients with clinically apparent UPE [4, 5]. Appropriate to the context, transesophageal echocardiography (TEE) assessment of heart and lungs can be particularly helpful in the immediate perioperative period for evaluating pulmonary and cardiac causes of hypoxemia simultaneously, as outlined in a review article by Cavayas *et al.* [7]. Even the specific transthoracic lung ultrasonography protocols routinely employed in screening for acute pulmonary edema, such as assessment of B-lines, can also come in handy by providing a non-invasive alternative for early identification and assessing patient response to therapy.

Interestingly, a recent 10-year follow up study by Puehler *et al.* observed that the consequences of UPE can range from decreased partial pressure of arterial oxygen/fraction of inspired oxygen ratio (PaO₂/FiO₂), increased blood loss, delayed extubation, escalated requirement of extracorporeal life-support, higher rate of tracheostomy and an increased length of intensive care unit stay [1]. Therefore,

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prevention strategies assume utmost importance given the significant impact of UPE on the surgical outcomes. Independent research groups have proposed modifications to the conventional protocols so as to mitigate the effects of ischemia-reperfusion such as: avoidance of preoperative vasodilators, cooling on CPB to 28–32°C, minimizing OLV time or avoiding OLV whenever surgically feasible, while maintaining a mean systemic pressure > 65 mm Hg on CPB to prevent deflated lung ischemia, thereby attenuating the incidence of UPE [1, 4]. It has also been found that use of neutrophil elastase inhibitors such as sivelestat significantly improved the alveolar-arterial oxygen difference and PaO₂/FiO₂ ratio in patients predisposed to UPE undergoing MICS [8].

However, once UPE sets in, it is managed akin to acute pulmonary edema bearing in mind its one-sided nature. Kitahara *et al.* successfully managed a case of severe UPE with veno-venous extracorporeal membrane oxygenation (V-V ECMO) along with differential lung ventilation utilizing low FiO₂, high positive end expiratory pressure (PEEP) and respiratory rate (60%, 20 mm H₂O and 5/min) on the affected side while maintaining high FiO₂, low PEEP and respiratory rate (100%, 10 mm H₂O and 18/min) on the normal side using a dual ventilator setup, along with a short course of pulse steroid therapy [9].

To conclude, amidst a growing popularity of MICS, it becomes imperative to acknowledge the peculiar future perioperative challenges likely to be posed by UPE. Our surgical and anesthesia protocols must therefore constantly evolve to address these intriguing complications emanating from novel surgical techniques. Further quality research in the area through large randomized controlled trials is needed given that the definitive etio-pathogenesis of the index problem continues to be elusive. Meanwhile, considering the potentially disastrous outcomes of this apparently uncommon complication, it is crucial to keep both eyes *maximally* open for signs of UPE in the era of *minimally* invasive surgical interventions.

Disclosure

The authors report no conflict of interest.

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