REVIEW ARTICLE

ATRIAL ARRHYTHMIAS FOLLOWING PULMONARY THROMBOENDARTERECTOMY: A COMPREHENSIVE REVIEW OF CURRENT LITERATURE

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Abstract

Chronic thromboembolic pulmonary hypertension (CTEPH) presents as a progressive vascular condition arising from previous episodes of acute pulmonary embolism, contributing to the development of pulmonary hypertension (PH). Pulmonary thromboendarterectomy (PTE) is the gold-standard surgical treatment for CTEPH; however, it may be associated with postoperative sequelae, including atrial arrhythmias (AAs). This comprehensive literature review explores the potential mechanisms for PTE-induced AAs with emphasis on the role of PH-related atrial remodelling and the predisposing factors. The identified preoperative predictors for AAs include advanced age, male gender, elevated resting heart rate, previous AAs, and baseline elevated right atrial pressure. Furthermore, we explore the available data on the association between post-PTE pericardial effusions and the development of AAs. Lastly, we briefly discuss the emerging role of radiomic analysis of epicardial adipose tissue as an imaging biomarker for predicting AAs.

Keywords: *Atrial arrhythmias, postoperative atrial fibrillation, chronic thromboembolic pulmonary hypertension, pulmonary thromboendarterectomy, narrative review.*

INTRODUCTION

Chronic thromboembolic pulmonary hypertension (CTEPH), which falls into group 4 of the World Health Organization's clinical classifications of pulmonary hypertension (PH)[1], is an atypical, progressive pulmonary vascular condition induced by a prior acute pulmonary embolism (PE). While the precise figures regarding its prevalence and yearly occurrence remain uncertain, research indicates that CTEPH has gradually increased in recent years and may manifest in approximately 4-5% of patients following an acute PE, placing it among the leading causes for pre-capillary PH[2,3].

Clinically, the hallmark signs of CTEPH are chronic, fibrotic scar tissue-like stenosis accompanied by persistent narrowing of the proximal pulmonary arteries due to obstructive intraluminal organized thromboembolic material[4]. These pathological events result in secondary vascular remodelling of the pulmonary microvascular bed, elevated pulmonary vascular resistance, and progressive right ventricular failure[4].

The long-term outcomes of CTEPH are typically unfavourable without surgical intervention[5]. Thus, pulmonary thromboendarterectomy (PTE) has become the gold-standard

Fig 1. Potential Mechanisms for Atrial Arrythmias After Pulmonary Thromboendarterectomy

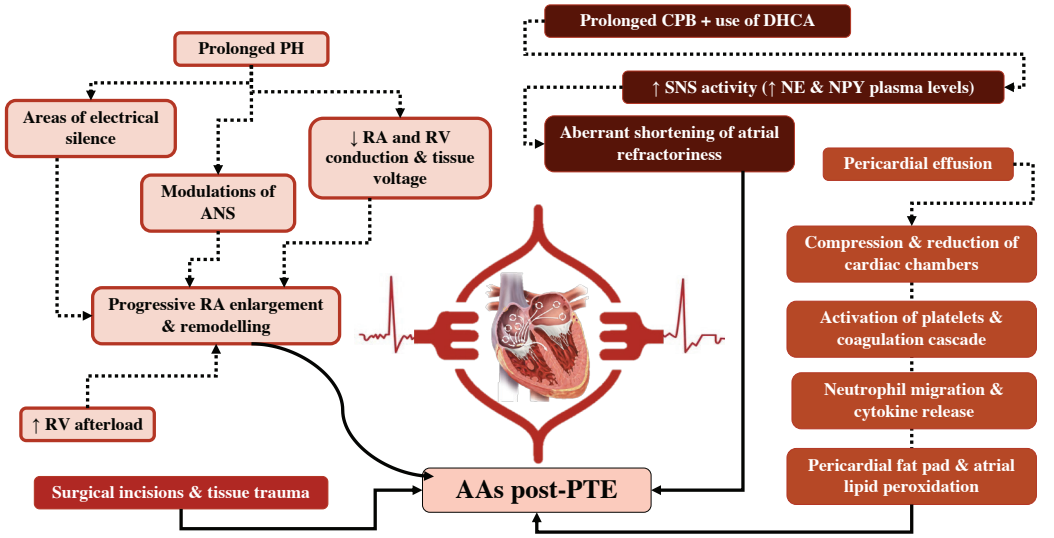

Figure 1

Visual representations of the proposed mechanisms for atrial arrythmias after pulmonary thromboendarterectomy reported in the literature (Abbreviations – AAs: atrial arrythmias, ANS: autonomic nervous system, CPB: cardiopulmonary bypass, DHCA: deep hypothermic circulatory arrest, NE: norepinephrine, NPY: neuropeptide Y, PH: pulmonary hypertension, PTE: pulmonary thromboendarterectomy, RA: right atrial, RV: right ventricular, SNS: sympathetic nervous system).

treatment choice over performing a balloon pulmonary angioplasty since its introduction in 1970[6]. The efficacy of PTE has been thoroughly demonstrated, with several case series from numerous institutions, including a multicenter registry study in China, where it was found to be an independent predictor of survival, with a 5-year survival rate of 87.5% in surgical patients[7].

Additionally, PTE is known to be technically challenging as it necessitates specialized training and advanced post-operative critical care[2,8]. It generally involves a complete endarterectomy, completed using a median sternotomy and supplemented by cardiopulmonary bypass and periods of deep hypothermia with circulatory arrest (DHCA)[2,4,8]. Nevertheless, with thorough patient selection, surgical practice and post-operative supervision, PTE can potentially induce significant pulmonary hemodynamic (improved cardiac index and reduced pulmonary vascular resistance) and clinical improvements. Mechanistically, this procedure ameliorates pulmonary ventilation–perfusion mismatch, thereby reducing right ventricular (RV) dysfunction and tricuspid regurgitation, inhibiting arteriopathic modifications in the small pulmonary vessels and preventing retrograde expansion of thromboembolic material[2].

Incidence of Atrial Arrhythmias after PTE

Postoperative AAs, particularly, postoperative atrial fibrillation (POAF), are one of the most frequent complications in patients undergoing PTE, with a reported 10–24.8% incidence rate[9–15]. Furthermore, this incidence is consistently higher in males compared to females in the literature, with rate of 52.4-73% and 27-47.6%, respectively[12–14]. It has been associated with longer intensive care and hospital length of stay, reduced cardiac functional capacity, higher resource utilization and increased morbidity and mortality[12,13]. Moreover, these tachycardias have been shown to be a predictor that adversely impacts the health-related quality of life (QOL) of patients a year after PTE[11].

Potential mechanisms for PTE-induced AAs

In the study by Farasat et al, authors aimed at identifying the potential risk factors for developing AF and atrial flutter after PTE[12]. Their consideration for an AA being present was based on telemetry findings and whether the supraventricular tachycardia required intervention [12]. Authors point out that most patients who undergo PTE are diagnosed with PH, a known predictor for the development of AAs[12]. This suggestion is endorsed by the findings of the study by Havranek et al, who elaborated on the role of PH and elevated afterload of the right ventricle (RV) on the progressive enlargement of the right atrium (RA)[13,16]. These pathological modifications, along with the fact that chronic PH leads to modulations of the autonomic system, areas of electrical silence and a reduction in conduction and tissue voltage in the RA and RV, may initiate and sustain AAs[17–19].

Moreover, the authors proposed the role of surgical incisions and tissue trauma on amplifying the risk of post-operative AAs[13]. The authors concluded that the progressive right atrial remodelling may also play a pivotal role in arrhythmogenesis following PTE[13]. These links between PH and AA were further recognized in Olsson et al.'s five-year prospective study of PH patients, in which they identified a 25.1% aggregate 5-year incidence of new-onset AAs[20].

In addition, the study by Farasat et al suggested that prolonged CPB times and the use of circulatory arrest under deep hypothermia may also play a role in increasing the risk of

postoperative AAs this group of patients[21,22]. This suggestion is endorsed by the finding that more profound degrees of hypothermia have been linked to amplified sympathetic nervous system (SNS) activity, as evidenced by a rise in norepinephrine and neuropeptide Y plasma levels during the rewarming period of CPB[23,24]. Furthermore, this aberrant stimulation of the SNS may trigger diverse alterations with the potential to induce and propagate AAs, including the shortening of atrial refractoriness in an aberrant manner[25,26].

Lastly, several studies have also suggested that postoperative pericardial effusion may play an indirect role in the development of POAF after cardiac surgery[27–29]. In a retrospective study of 502 patients by Zhang et al, the incidence of pericardial effusion post-PTE was about 24.7%, with the most predictive risk factors being younger age, male sex, and re-entry sternotomy[27]. However, other studies have reported a rate of 6.7-19%, highlighting a broad variance in occurrence[12,14,30]. Pericardial effusion is thought to trigger POAF by causing acute compression and acute reduction in size of cardiac chambers[29,31]. In addition, there is growing evidence that residual pericardial blood collection is associated with various pro-inflammatory changes that result in the activation of platelets and the coagulation cascade, which, in turn, can trigger neutrophil migration and cytokine release (IL-6, IL-8, IL-1)[32–34]. Furthermore, neutrophil-generated peroxide initiates the formation of reactive oxygen species, culminating in the oxidative stress pathway's endpoint: lipid peroxidation of both the pericardial fat pads and the atria thus increasing the risk for POAF[35]. These aforementioned potential mechanisms are summarized below in Figure 1.

Predictors for Developing AAs following PTE

In the study by Farasat et al, multivariate logistic regression analysis showed that the strongest risk factors for developing AAs after PTE were previous history of AA, advanced age, and male sex, while preoperative RA pressure showed a marginally significant predictive effect[12]. Regarding gender, some have postulated that females possess a sex-specific, POAF-protective, hormone-related mechanism, however this has yet to be confirmed by robust investigations[36]. As for advanced age, this powerful predictor has long been established as a risk factor for cardiac surgery, including PTE[37]. Not only do older adult patients accumulate conventional risk factors associated with AAs over their lifetime, but they also experience aberrant age-related atrial myocardium remodelling[38–40]. Furthermore, as individuals age, the reduced flexibility of the LV results in LA expansion along with higher filling pressures (in response to volume load) that contribute to atrial stretch[38]. Collectively, these aged-related changes generate a more conducive environment for the development of AAs[38].

Havranek et al's study identified similar risk factors however, they also reported that patients who developed AAs more frequently had arterial hypertension and were in a worse New York Heart Association (NYHA) class[13]. Regarding arterial hypertension, this factor has been long established as a powerful predictor of AAs that is not exclusive to those undergoing PTE[41]. Extensive research suggests that arterial hypertension triggers hemodynamic changes such as reduced left ventricular (LV) diastolic function and rise in LV stiffness and wall thickness[41]. Subsequently, these events result in an increase in left atrial (LA) stretch and pressure that, in turn, induces a state of atrial cardiomyopathy in the hypertrophied heart[41]. This is marked by a series of complex structural, architectural, contractile, and electrophysiological atrial modifications predisposing individuals to AAs, namely POAF[41].

Also, a worse NYHA class indicates a more aggressive form of heart failure (HF), a recognized cause and consequence of AAs[42,43]. Specifically, patients with worsening HF are at

Table 1 **The incidence and risk factors associated with AAs post-PTE.**

Author (date)	Incidence of AAs	Statistically Significant Perioperative Risk Factors
Havranek et al. (2020)	29%	Advanced age Prior arterial hypertension Worse NYHA class
Farasat et al. (2019)	24.2%	Advanced age Male sex Prior incidence of AAs Baseline right atrial pressure Longer CPB time Concomitant CABG
Liu et al. (2022)	21.6%	Advanced age Higher resting heart rate Higher platelet count

Abbreviations: AAs - atrial arrythmias, CABG - Coronary Artery Bypass Graft, NYHA - New York Heart Association.

elevated risk of POAF, especially after cardiac operations such as PTE[43]. It is widely known that HF can lead to increased cardiac filling pressures, autonomic and neuroendocrine dysfunction, and dysregulation of intracellular Ca^{2+} levels[43]. Additionally, HF is linked with amplified interstitial fibrosis, a substrate for abnormal atrial conduction, thereby establishing a foundation for AAs, primarily in animal models[44].

Finally, a retrospective study by Liu et al aimed at assessing the predictive value of preoperative resting heart rate (RHR) for POAF in the context of PTE once again found advanced age to be an independent risk factor alongside RHR after completing a binary logistic regression analysis[14]. While the underlying mechanism behind RHR as a risk factor is not fully elucidated, the authors provide two explanations. Firstly, a higher RHR signifies a high baseline sympathetic tone, which, as explained in the above sections, plays a role in arrhythmogenesis[14]. The secondary explanation is that reduced diastolic filling time (a consequence of a condensed cardiac cycle) leads to myocardial hypoxia, which has been shown to influence the initiation and propagation of AAs[14,45]. These findings are encapsulated in the Table 1 below.

Radiomics and Predicting Risk of AAs Following PTE

Despite the identification of the risk factors mentioned above, some authors believe that clinical features alone may provide limited usefulness in predicting AAs after PTE[15]. Thus, for a more individualized assessment and prediction of the risk of AAs after PTE, Liu et al proposed using the radiomic signature of epicardial adipose tissue (EAT) as an emerging biomarker[15]. After reviewing the preoperative computed tomography pulmonary angiography images of CTEPH patients who underwent PTE, the authors selected 5 of the 1,218 radiomics features to construct the radiomics signature[15]. This signature ultimately showed good discrimination, calibration, and clinical practicability[15].

This in fact correlates with the findings that EAT can produce several pro-inflammatory adipokines, such as activin-A and interleukin-1b, that are well-known for triggering the onset of POAF[46–48]. EAT volume and radiodensity, as quantified by cardiac computed tomography (CT), have also reportedly been linked with the incidence, severity, and recurrence of AAs[49–52]. Therefore, the employment of radiomic analysis, as proposed by the authors, can add a new dimension to the screening of POAFs by enabling clinicians to extract more data based on more accurate and comprehensive interpretation of tissue imaging[15].

CONCLUSION

Postoperative atrial arrhythmias occur frequently after PTE and may be associated with increased morbidity and resource utilization. Several mechanisms and clinical risk factors have been associated with the development of atrial arrhythmias in these patients. The development of a predictive model that incorporates clinical, radiological, and laboratory parameters may help reduce the clinical burden of this condition.

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