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CARDIAC ARRHYTHMIAS IN PULMONARY HYPERTENSION: A REVIEW OF MECHANISMS, PREVALENCE, CLINICAL SIGNIFICANCE, AND PATIENT FUNCTIONAL CAPACITY

Adam Rafałowicz, [AR]
University Clinical Hospital in Bialystok,
ul. M. C. Skłodowskiej 24a, 15-276 Białystok, Poland
https://orcid.org/0009-0005-2535-6884
e-mail: adam.rafalowicz3321@gmail.com

Urszula Justyna Wojciechowska, [UW]
Medical University of Bialystok,
ul. Jana Kilińskiego 1, 15-089 Białystok, Poland
https://orcid.org/0009-0006-8800-2386
e-mail: wojciechowskaula26@gmail.com

Jakub Mierzejewski, [JM]
Masovian Specialist Hospital named after Dr Józef Psarski in Ostrołęka,
ul. aleja Jana Pawła II 120A, 07-410 Ostrołęka
https://orcid.org/0009-0004-6689-8746
e-mail: jakub.mierzejewski99@gmail.com

Bartosz Łuniewski, [BL]
Medical University of Bialystok,
ul. Jana Kilińskiego 1, 15-089 Białystok, Poland
https://orcid.org/0009-0005-8934-6626
e-mail: bartosz.lun12@gmail.com

Angelika Macko, [AM]
Medical University of Bialystok,
ul. Jana Kilińskiego 1, 15-089 Białystok, Poland
https://orcid.org/0009-0007-1274-2481
e-mail: angel1682000@gmail.com

Monika Olszańska, [MO]
Medical University of Bialystok,
ul. Jana Kilińskiego 1, 15-089 Białystok, Poland
https://orcid.org/0009-0006-2511-505X
email: moniolsza00@gmail.com

Kamil Klaudiusz Lauko, [KL]
Medical University of Bialystok,
ul. Jana Kilińskiego 1, 15-089 Białystok, Poland
https://orcid.org/0000-0003-0727-3661
e-mail: kamillauko.research@gmail.com

Aleksandra Sokół, [AS]
University Clinical Hospital in Bialystok,
ul. M. C. Skłodowskiej 24a, 15-276 Białystok, Poland
https://orcid.org/0009-0008-9707-9179
e-mail: ola.sokol24@gmail.com

Klaudia Mościszko, [KM]
University Clinical Hospital in Bialystok,
ul. M. C. Skłodowskiej 24a, 15-276 Białystok, Poland
https://orcid.org/0009-0001-4919-6898
e-mail: klaudiamosciszko@gmail.com

Dominika Hakało, [DH]
University Clinical Hospital in Bialystok,
ul. M. C. Skłodowskiej 24a, 15-276 Białystok, Poland
https://orcid.org/0009-0004-9363-8747
e-mail: d.hakalo@wp.pl

Abstract

Introduction:

Pulmonary hypertension is a chronic cardiovascular disease defined by a mean pulmonary artery pressure (mPAP) of > 20 mmHg, as measured by right heart catheterization (RHC). Rhythm disturbances are relatively common in this condition and significantly worsen patient prognosis. Elucidating the mechanisms that drive arrhythmia development may help identify therapeutic intervention strategies to enhance patient outcomes.

Materials and methods:

We performed a literature review of epidemiology, pathogenesis, and clinical impact of cardiac arrhythmias in Pulmonary Arterial Hypertension (PAH) and Chronic Thromboembolic Pulmonary Hypertension (CTEPH), analysing registry data, histopathological and electrophysiological studies, and clinical outcomes.

Results:

Supraventricular arrhythmias (SVA) occur in 10–33 % of patients, rising to ~30 % over 10 years. Key mechanisms

include atrial stretch and fibrosis, autonomic imbalance, and right ventricular ischemia. Arrhythmias correlate with reduced cardiac output, impaired exercise capacity, and increased mortality.

Conclusions:

Early detection and maintenance of sinus rhythm enhance survival and functional capacity, highlighting the necessity of research and interventions directed at atrial remodelling, neurohormonal modulation and myocardial ischaemia.

Keywords:

pulmonary hypertension; supraventricular arrhythmias; PAH; CTEPH; heart remodelling.

1. INTRODUCTION

Pulmonary hypertension (PH) is defined by a mean pulmonary artery pressure (mPAP) greater than 20 mm Hg at rest as determined by right heart catheterization and represents a progressive cardiovascular disorder that arises secondary to a wide array of clinical conditions, most frequently in the setting of underlying cardiac or pulmonary disease [1]. Patients with PH are stratified into five distinct clinical groups on the basis of predominant pathophysiological mechanisms, characteristic clinical presentations, specific hemodynamic profiles and targeted therapeutic strategies, in accordance with the 2022 guidelines issued by the European Society of Cardiology and the European Respiratory Society [1]. These five groups comprise pulmonary arterial hypertension (PAH), pulmonary hypertension due to left heart disease, pulmonary hypertension associated with pulmonary parenchymal disease or chronic hypoxia, pulmonary hypertension resulting from pulmonary artery obstruction (for example chronic thromboembolic pulmonary hypertension, CTEPH) and pulmonary hypertension with multifactorial or unclear mechanisms [1].

Pulmonary arterial hypertension (PAH) in particular is distinguished by progressive remodelling and luminal narrowing of the distal pulmonary vasculature, which gives rise to a sustained increase in pulmonary vascular resistance; this in turn imposes a chronic pressure overload upon the right ventricle, predisposing to right ventricular dysfunction, volume overload and ultimately death, with reported survival rates of approximately 92 percent at one year, 84 percent at two years and 79 percent at three years following diagnosis [2]. As pulmonary hypertension advances, the escalating hemodynamic burden on the right ventricle

often culminates in right ventricular failure and systemic venous congestion, which manifest clinically as peripheral edema, ascites, hepatomegaly and elevated jugular venous pressure [3]. Notably, arrhythmias, particularly supraventricular arrhythmias such as atrial flutter and atrial fibrillation, are recognized as common complications in patients with PAH and CTEPH [5]. Their development is closely linked to structural and functional remodelling of the right heart chambers, including dilatation, myocardial hypertrophy and interstitial fibrosis, as well as to neurohormonal and autonomic imbalances [4]. In particular, right atrial enlargement and chronically elevated atrial wall stress promote the deposition of fibrotic tissue and the creation of areas of heterogeneous conduction, thereby establishing an anatomical and electrophysiological substrate conducive to the initiation and maintenance of supraventricular arrhythmias [4,5]. The main aim of the present study is to present the current state of knowledge regarding the prevalence of cardiac arrhythmias in the course of this disease, their likely underlying causes, and the impact of rhythm disturbances on functional status and overall prognosis.

2. EPIDEMIOLOGY

It was shown in studies that approximately one in ten patients with pulmonary arterial hypertension (PAH) already exhibited heart rhythm disturbances at the time of diagnosis, most commonly atrial fibrillation (AF), followed by atrial flutter (AFL) or other supraventricular tachyarrhythmias [6]. The risk of developing new arrhythmias rises progressively over time, with about 7 percent of those with an initially normal rhythm developing arrhythmias within one year, nearly 20 percent by five years, and almost 30 percent by ten years, underscoring the need for long-term surveillance in this population [6]. Other research has demonstrated that atrial arrhythmias, including atrial fibrillation (AF) and atrial flutter (AFL), occur frequently in individuals diagnosed with pulmonary arterial hypertension (PAH) or chronic thromboembolic pulmonary hypertension (CTEPH), with incidence estimates ranging between 10 percent and 33 percent across different cohorts and clinical settings [5]. While many patients initially present with stable sinus rhythm, a significant proportion, up to one third, may eventually develop arrhythmic events, highlighting the importance of continuous rhythm monitoring and prompt therapeutic response to optimize clinical outcomes [5]. Additionally, the likelihood of arrhythmia recurrence in this group remains substantial, with some studies indicating recurrence rates as high as 46 percent and reported values ranging from 15 percent to 64 percent across various patient populations and follow-up periods [5].

3. PATHOGENESIS

Given the relatively limited research dedicated specifically right-sided heart remodelling in the context of pulmonary hypertension, our current understanding of the complex pathophysiological mechanisms driving cardiac arrhythmias in this setting remains incomplete and continues to challenge both clinicians and investigators [28]. This persistent knowledge gap likely reflects a multifactorial etiology in which chronic hemodynamic overload, characterized by sustained elevated pulmonary artery pressures and wall stress, triggers a cascade of compensatory and maladaptive myocardial structural changes, including chamber dilatation, fibrosis and altered extracellular matrix composition that progressively impair contractile function and electrical stability, while persistent neurohormonal activation such as sympathetic nervous system upregulation and oxidative stress induces further adverse modifications in myocardial histology and electrical conduction pathways, and intricate molecular and electrophysiological alterations at the ion channel and gap junction levels, encompassing altered calcium handling, action potential duration changes and connexin expression modifications, operate within an elaborate network of reciprocal interactions to ultimately promote arrhythmogenesis in pulmonary hypertension [4,9,8,10].

3.1 HEART REMODELING

Elevated pulmonary vascular resistance in pulmonary hypertension imposes a substantially increased afterload on the right ventricle, which in turn elevates myocardial wall stress and initiates a cascade of maladaptive responses. Initially, compensatory hypertrophy of right ventricular cardiomyocytes occurs to normalize wall tension; however, persistent pressure overload eventually overwhelms these adaptive mechanisms, leading to chamber dilation and systolic dysfunction typical of right heart failure [11,12]. As right ventricular end-diastolic pressure rises, this hemodynamic burden is transmitted retrogradely to the right atrium, resulting in an increase in right atrial pressure and progressive chamber enlargement—key hallmarks of advanced pulmonary hypertensive disease [12]. Right atrial enlargement has been robustly linked to a heightened propensity for supraventricular arrhythmias, most notably atrial fibrillation. Multiple clinical studies have demonstrated that patients exhibiting larger right atrial dimensions experience a significantly greater incidence of SVAs compared to those with normal atrial size [13,14]. Beyond purely geometric remodelling, pathological alterations in the atrial extracellular matrix (ECM) and cell populations play a pivotal role in arrhythmogenesis.

Chronic hemodynamic stress and neurohormonal activation drive resident cardiac fibroblasts to transdifferentiate into myofibroblasts, which secrete excessive amounts of fibrillar collagen and other ECM proteins [15]. This fibrotic expansion increases tissue stiffness, disrupts the homogeneity of electrical conduction by introducing areas of slowed impulse propagation, and fosters the formation of re-entrant circuits—central mechanisms underpinning atrial fibrillation [5]. In addition, fibroblasts and myofibroblasts can electrically couple with adjacent cardiomyocytes via connexin-mediated gap junctions; owing to their relatively depolarized resting membrane potential, these nonmyocytic cells may act as ectopic pacemakers, further facilitating both the initiation and maintenance of atrial fibrillation [15]. Histopathological examination of right atrial biopsies from patients with chronic thromboembolic pulmonary hypertension (CTEPH) reveals pronounced cardiomyocyte hypertrophy that is inadequately matched by capillary angiogenesis [7]. Although the absolute number of capillaries per cardiomyocyte increases, the proportional thickening of myocytes outpaces vascular proliferation, culminating in a net reduction of capillary density per unit tissue area and predisposing to relative myocardial ischaemia [7,29]. This microvascular insufficiency likely contributes to the arrhythmogenic substrate [29]. In a separate electrophysiological study examining chronic pulmonary hypertension's impact on atrial substrate, idiopathic pulmonary arterial hypertension was associated with marked right atrial conduction slowing, reduced signal amplitude with expanded low-voltage areas, the emergence of electrically silent regions, impaired sinus-node function, and an increased susceptibility to atrial fibrillation. These alterations establish a pathophysiological substrate for arrhythmogenesis and explain the high prevalence of supraventricular rhythm disturbances in chronic pulmonary hypertension [16].

3.2 AUTONOMIC NERVOUS SYSTEM

In addition to structural and electrophysiological remodelling, augmented sympathetic nervous system activity represents a key modulatory factor in arrhythmogenesis within pulmonary hypertension [4]. Microneurographic assessments have demonstrated that patients with pulmonary arterial hypertension exhibit significantly elevated muscle sympathetic nerve activity compared with matched healthy individuals, indicating chronic sympathetic overdrive in this population [17]. Concordantly, circulating levels of norepinephrine are increased in pulmonary hypertensive patients and show a positive correlation with pulmonary vascular resistance, a central determinant of disease severity, suggesting that heightened adrenergic tone both reflects and exacerbates right-sided hemodynamic compromise [18,19].

3.3 RIGHT VENTRICLE ISHAEMIA

Although ventricular arrhythmias are relatively infrequent in pulmonary hypertension, their occurrence carries a disproportionately high risk compared to supraventricular rhythm disturbances [6]. These malignant ventricular rhythm disturbances can precipitate sudden cardiac death, which is responsible for approximately 28 % of fatalities in patients with pulmonary arterial hypertension [20,21]. Among the myriad factors predisposing to ventricular arrhythmogenesis, myocardial ischemia in the hypertrophied ventricle can create a substrate for ischemia-induced ventricular and tachyarrhythmias [21,24,29]. Experimental models of right ventricular failure lend insight into this process. In rodent models subjected to chronic pressure overload, excessive generation of reactive oxygen species (ROS) within the right ventricular myocardium has been documented, concomitant with an inadequate upregulation of intrinsic antioxidant defenses [22,23]. Other studies have demonstrated the deleterious effects of reactive oxygen species on the myocardium, including induction of arrhythmias, contractile dysfunction and adverse remodelling [23].

4. CLINICAL SIGNIFICANCE

Effective atrial contraction contributes significantly to ventricular filling and is essential for preserving stroke volume [25]. During atrial fibrillation, the loss of organized atrial systole reduces stroke volume by approximately 20–30%, underscoring the hemodynamic impact of atrial dysrhythmia [25]. Beyond its mechanical consequences, the onset of supraventricular arrhythmias in PAH patients correlates with measurable clinical decline. In a cohort study of individuals with pulmonary arterial hypertension, those who developed supraventricular arrhythmias experienced a marked worsening of right-heart failure symptoms and overall functional status; moreover, persistent atrial fibrillation was independently associated with increased mortality compared to patients remaining in sinus rhythm [26]. Further evidence comes from a comparative analysis in which PAH patients with atrial fibrillation were matched against counterparts in stable sinus rhythm. Assessments using NYHA/WHO functional class, six-minute walk distance, and NT-proBNP concentrations revealed that the atrial fibrillation group not only demonstrated significantly poorer exercise tolerance but also bore a higher functional class burden [27]. Collectively, these findings highlight the clinical advantage of maintaining sinus rhythm: conversion back to organized atrial activity in PAH and CTEPH populations has been linked to enhanced survival outcomes and improved exercise capacity, reinforcing the therapeutic value of rhythm control strategies in this high-risk cohort [5].

5. SUMMARY AND CONCLUSION

Cardiac rhythm disturbances in pulmonary hypertension—particularly in patients with PAH and CTEPH, pose a formidable clinical challenge due to their multifactorial origin. Key drivers include structural remodelling (notably right-atrium dilation), cellular adaptations such as cardiomyocyte hypertrophy with insufficient capillary growth, and microenvironmental alterations encompassing fibrosis, ion-channel dysfunction, and autonomic imbalance. An ischemic substrate, created by impaired myocardial perfusion and oxidative stress, further amplifies arrhythmogenic potential. The deleterious effects of these arrhythmias on exercise capacity, right-heart function, and survival are well established, as is the clear benefit of restoring and maintaining sinus rhythm. The adverse impact of arrhythmias on functional status and survival in pulmonary-hypertension patients has been well documented, as have the benefits of restoring sinus rhythm. Future research should focus on mechanistic studies of right-atrial electrophysiology, development of risk-stratification tools for arrhythmia onset, and targeted therapies addressing fibrotic and neurohormonal pathways.

Disclosure

Author's contribution

Conceptualization: **AR**

Methodology: **UW, KL, AR**

Formal analysis: **MO, AS, DH,**

Investigation: **AS, UW, AM, AS, MO,**

Writing-rough preparation: **DH, AR, KM, JM, BL**

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