Review

Right ventricular strain: Cardiovascular challenges in pulmonary diseases

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ABSTRACT
Right heart failure is associated with a poor prognosis, and right ventricular (RV) strain, assessed through echocardiography, is a valuable method for evaluating right heart function. However, discussions on this topic remain limited. This article explores RV strain induced by pulmonary diseases to increase awareness of how these conditions can exacerbate right heart failure. Pulmonary embolism leads to increased RV afterload, resulting in RV dilation and ischemia, which can progress to cardiogenic shock. Echocardiography is effective in detecting RV strain and assessing the severity of pulmonary embolism. In Chronic Obstructive Pulmonary Disease (COPD), increased pulmonary vascular resistance causes RV dysfunction, identifiable through speckle-tracking echocardiography (STE). Pulmonary fibrosis may contribute to right heart failure through pulmonary hypertension. RV Longitudinal Strain (RVLS) is an important prognostic marker in patients with pulmonary hypertension and COVID-19, where low RVLS (≤20.5%) is associated with higher mortality. In conclusion, right heart failure carries a poor prognosis, and RV strain evaluation using STE is a useful tool for early detection. Pulmonary diseases, including COPD, pulmonary embolism, pulmonary fibrosis, pulmonary hypertension, and COVID-19, can induce RV strain, which is critical for assessing prognosis and guiding disease management.

KEYWORDS: Right ventricular strain; cardiovascular diseases; pulmonary diseases; right heart failure.

INTRODUCTION

Heart failure is a serious medical condition where the heart is unable to pump blood efficiently, leading to reduced oxygen and nutrient delivery to various organs and tissues. It is generally classified into two primary categories: right-sided heart failure and left-sided heart failure. While both types can significantly impact a patient's quality of life, right-sided heart failure tends to have a worse prognosis compared to left-sided heart failure.[1] The survival rates are strikingly different between the two. A two-year survival rate for individuals with biventricular heart failure (both right and left heart failure) is as low as 23%, in stark contrast to the 71% survival rate for individuals who primarily suffer from left-sided heart failure.[2] The CHARITEM study, which examined heart failure cases across multiple regions, found that right-sided heart failure represents about 2.2% of all heart failure cases and serves as a secondary cause of left-sided heart failure in over 20% of such instances.[3] The prevalence of right-sided heart failure can vary significantly by geographic region. For instance, data from the European Society of Cardiology's Egyptian registry reveals that right-sided heart failure is present in 4.5% of acute heart failure cases.[4]

Diagnosing right-sided heart failure involves several diagnostic methods, with echocardiography being one of the most widely used due to its ability to provide quick, non-invasive assessment. One crucial parameter measured during echocardiography is the right ventricular (RV) strain, which plays a vital role in assessing right heart function.[5] RV strain evaluates both the workload on the right ventricle and any dysfunction that may be present. Abnormal patterns of RV strain are often indicative of disease progression, increased treatment demands, and a heightened risk of mortality in patients with various pulmonary conditions, including pulmonary arterial hypertension.[6] This article seeks to investigate RV strain caused by pulmonary diseases in order to raise awareness of how these conditions can worsen right heart failure.

RIGHT VENTRICULAR STRAIN AND LUNG DISEASES

Lung diseases have long been known to contribute to the development of right-sided heart failure, which is sometimes referred to as cor pulmonale. Right heart changes can occur acutely, as in cases of pulmonary embolism, or chronically, as seen in conditions such as Chronic Obstructive Pulmonary Disease (COPD).[7] The onset of right-sided heart failure in COPD is typically more gradual, occurring over a longer time period.[3] The purpose of this literature review is to explore how RV strain is implicated in lung diseases, highlighting how pulmonary conditions can exacerbate or contribute to the development of right-sided heart failure.

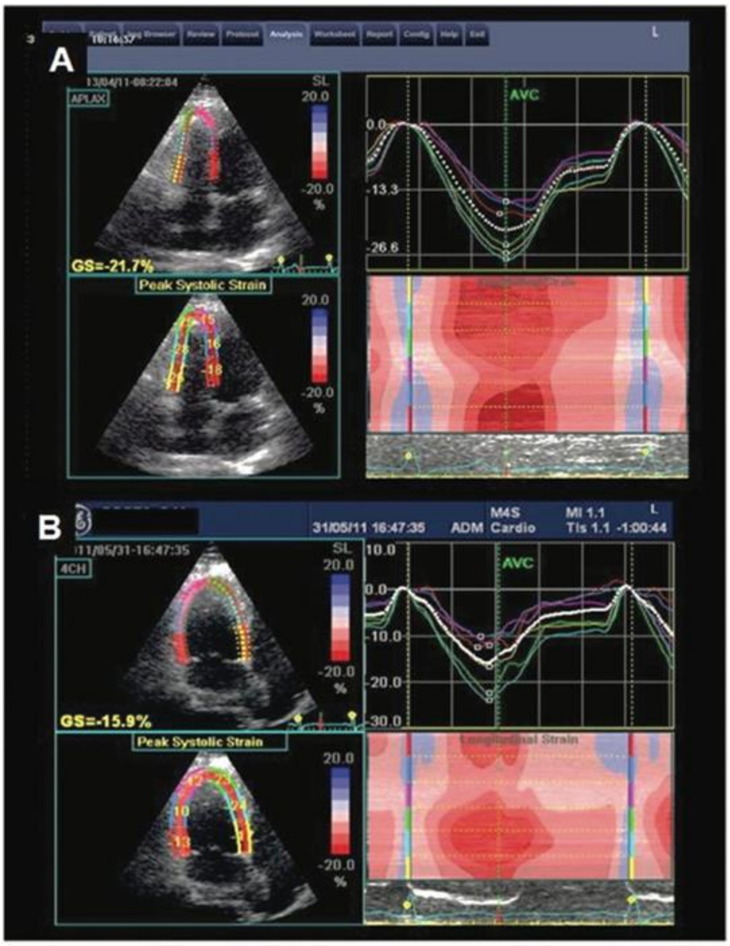

Figure 1. 2D speckle tracking analysis from a right ventricle-focused apical four-chamber view. The global longitudinal strain (GLS) and time curves were derived by tracking a six-segment

region of interest (ROI). In Patient A from the control group, the GLS is -21.7%, while in Patient B from the COPD group, the GLS is -15.9%.

Right ventricular strain and pulmonary embolism

In pulmonary embolism, RV afterload increases significantly. This occurs as a result of pulmonary artery pressure rising once a substantial proportion of pulmonary vessels (around 25-30%) becomes obstructed by the embolism.[8] In patients without pre-existing cardiopulmonary conditions, the right ventricle initially compensates for this increased afterload.[9] However, when more than 50-75% of the pulmonary vessels are blocked by the embolism, pulmonary artery pressure can rise above 40 mmHg. This is often exacerbated by hypoxia, which triggers local vasoconstriction through the release of vasoactive mediators like serotonin, thromboxane, and histamine.[10]

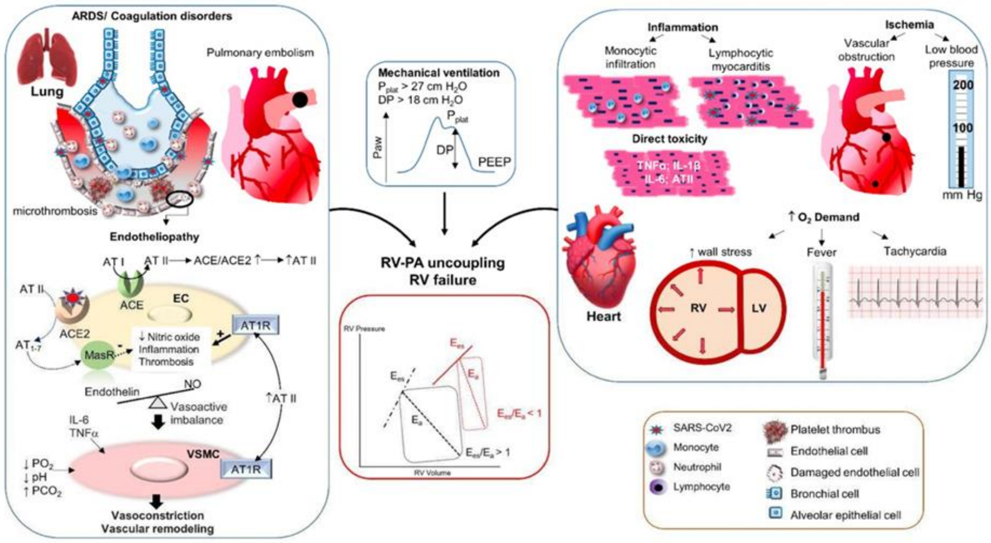

Figure 2. Pathophysiology of right ventricular (RV) dysfunction in COVID-19 ARDS. The diagram illustrates key mechanisms, including hypoxic pulmonary vasoconstriction, increased pulmonary vascular resistance, RV pressure overload, and reduced RV perfusion. The cascade leads to RV dilation, impaired systolic function, and hemodynamic compromise, exacerbating ARDS severity.

Once the afterload exceeds critical thresholds, the right ventricle may dilate in an attempt to compensate. This causes the left ventricle to become underfilled and leads to reduced coronary artery perfusion.[11] Blood flow to the right ventricle is diminished as a result of reduced coronary artery output and the increased intramuscular pressure that impedes coronary artery flow, resulting in ischemia of the right ventricle. As ischemia progresses, the contractility of the right ventricle declines, exacerbating RV dilation, reducing left ventricular output, and worsening hemodynamic instability. This cycle can eventually lead to cardiogenic shock.[12]

In suspected cases of pulmonary embolism, echocardiographic signs of RV strain include an increased right ventricle-to-left ventricle ratio, abnormal septal motion, McConnell's sign, tricuspid regurgitation, elevated pulmonary artery systolic pressure, and reduced tricuspid annular plane systolic excursion (TAPSE).[13] Additionally, reduced RV free wall strain measured via speckle tracking can help identify the severity of strain.[14] Longitudinal strain, which measures the systolic shortening of the right ventricular free wall from base to apex, is particularly helpful. Normal quantitative parameters vary with ultrasound equipment and software, but the American Society of Echocardiography (ASE) defines the average RV free wall strain as -29% ± 4.5%, with an abnormal value being less than -20% (or an absolute value

<20%).[15] In cases of pulmonary embolism, a decrease in RV strain is strongly correlated with poor in-hospital outcomes, including mortality and the need for additional treatments, such as thrombolysis or pulmonary artery thrombectomy.[16]

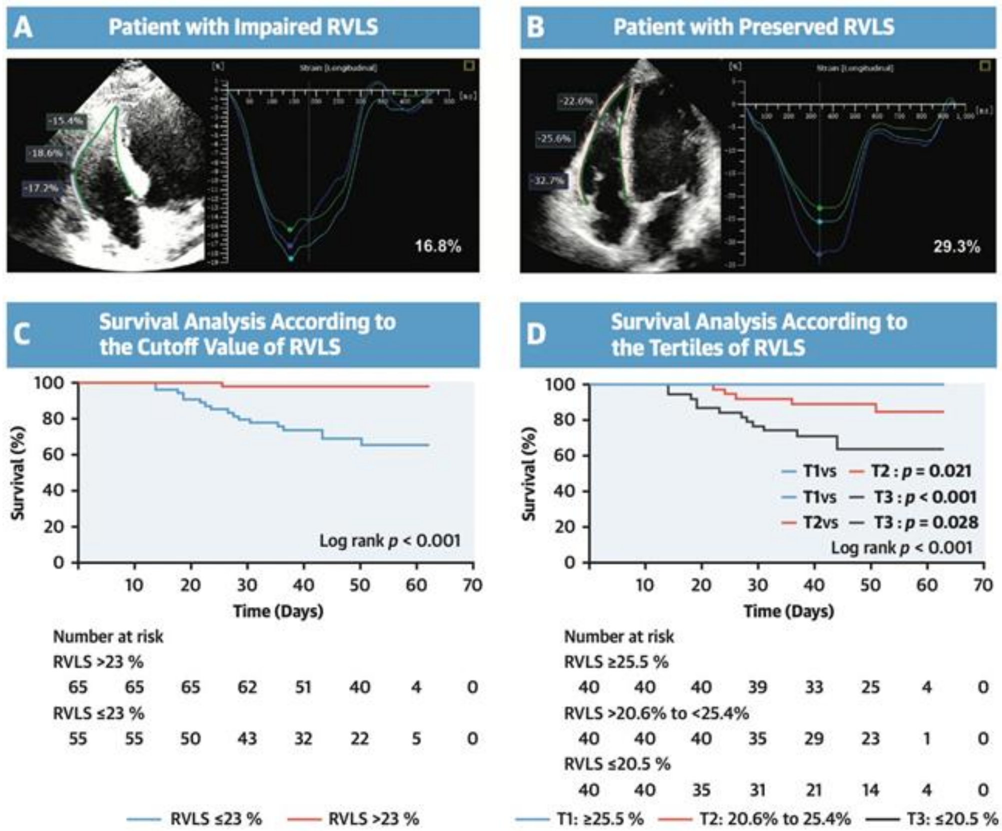

Figure 3. (A) A 42-year-old patient with impaired right ventricular longitudinal strain (RVLS) of 16.8%, who succumbed to COVID-19 17 days after echocardiography. (B) A 57-year-old patient with RVLS of 29.3%, who survived beyond a 61-day follow-up period. In the lower panels, survival curves for all-cause mortality are presented: (C) based on the RVLS cutoff value (23%) and (D) categorized by RVLS tertiles (≤20.5%, 20.6% to 25.4%, and ≥25.5%). RVLS values are expressed as absolute values.

Right ventricular strain and chronic obstructive pulmonary disease (COPD)

COPD is characterized by chronic hypoxemia and pulmonary vascular changes that result from the loss of parenchymal tissue and fibrosis. These factors lead to a gradual increase in pulmonary vascular resistance, typically around 3 mmHg per year, allowing sufficient time for compensatory mechanisms to kick in.[17] This chronic hypoxia can be worsened by hypercapnia and acidemia, which are common in advanced COPD. In addition, pulmonary hyperinflation in COPD patients increases right atrial pressure, which reduces venous return and, consequently, reduces the right ventricular preload. This effect contributes to a decline in RV function.[18]

To counteract the increased intraluminal pressure, the right ventricle undergoes hypertrophy. This adaptive process helps to reduce wall stress, as the thickening of the RV wall is accompanied by myocyte hypertrophy, remodeling of the myocardial extracellular matrix, and increased capillary density.[19] The development of concentric RV hypertrophy is commonly observed in stable COPD patients, and postmortem studies have found this condition in 76% of patients with advanced COPD. RV remodeling begins early, even before the onset of pulmonary hypertension, indicating the importance of evaluating RV function from the early stages of COPD.[20] Research by Botelho et al. in 2022 revealed that global longitudinal strain values differed significantly between control and COPD groups, further emphasizing the relevance of RV strain in these patients (Figure 1).[21]

A study by Nugroho et al., which examined 59 COPD patients at Harapan Kita Hospital, found that global strain values were higher in COPD patients without pulmonary hypertension compared to those with pulmonary hypertension, though this difference was not statistically significant (p=0.09).[22] However, a study by Kanar et al. demonstrated that speckle tracking echocardiography (STE) was more sensitive than conventional 2D echocardiography in detecting RV changes following a pulmonary rehabilitation program. Changes in RV free wall strain and global longitudinal strain before and after rehabilitation were significant, further confirming that RV strain is closely linked with functional improvement in COPD patients.[23]

Right ventricular strain and pulmonary fibrosis

Pulmonary fibrosis, which leads to progressive parenchymal scarring and pulmonary hypertension, often results in significant morbidity and mortality. Pulmonary hypertension in pulmonary fibrosis arises from several mechanisms, including hypoxia, smooth muscle hypertrophy, and collagen accumulation within the pulmonary arteries.[24] These changes contribute to the progression of pulmonary vascular disease and eventually lead to right-sided heart failure.[7]

D'Andrea et al. conducted a study in 55 patients with idiopathic pulmonary fibrosis (IPF), using STE to assess RV strain. Despite the absence of pulmonary hypertension in many of these patients, significant RV strain was detected, illustrating that right heart dysfunction occurs early in the course of pulmonary fibrosis. The study highlighted the importance of monitoring RV function even before the development of overt pulmonary hypertension.[25]

Right ventricular strain and pulmonary hypertension

Pulmonary hypertension is characterized by increased pulmonary arterial pressure, typically defined as a mean pulmonary artery pressure (mPAP) exceeding 20 mmHg. Pulmonary hypertension can arise due to a variety of conditions, particularly in lung diseases such as COPD and interstitial lung disease.[26] In patients with pulmonary hypertension, the remodeling of pulmonary vessels, including intimal thickening, smooth muscle hypertrophy, and loss of arteriolar density, contributes to increased vascular resistance and right ventricular strain.[27]

A meta-analysis by Hulshof et al., which reviewed 11 studies with a total of 1,169 patients, found that right ventricular longitudinal strain (RVLS) provides independent prognostic value in patients with pulmonary hypertension. RVLS was found to predict clinical events, such as hospitalization, need for lung transplantation, and intensive care admission, as well as overall mortality. These findings underscore the utility of RV strain as a critical prognostic tool in managing pulmonary hypertension.[28]

Right ventricular strain and COVID-19

The COVID-19 pandemic introduced a novel challenge for healthcare providers, with many patients suffering from severe respiratory distress, leading to right ventricular dysfunction. The pathophysiology behind RV dysfunction in COVID-19-related acute respiratory distress syndrome (ARDS) is multifaceted (Figure 2).[29] The viral infection triggers an inflammatory cascade that leads to endothelial damage, thrombosis, and vasoconstriction, further exacerbating pulmonary hypertension.[30] Additionally, mechanical ventilation in severe cases can worsen RV function by increasing pulmonary vascular resistance.[31]

A study by Li et al. found that RVLS was a strong predictor of mortality in COVID-19 patients. Patients with lower RVLS values had significantly higher mortality rates compared to those with higher RVLS values, underscoring the importance of RV strain assessment in predicting outcomes in COVID-19 (Figure 3).[32]

CONCLUSION

Right-sided heart failure is associated with poor clinical outcomes, and the assessment of RV strain through techniques like STE is invaluable in predicting prognosis and managing treatment. Various pulmonary diseases, including COPD, pulmonary embolism, pulmonary fibrosis, and pulmonary hypertension, can significantly contribute to RV strain. Early detection of RV strain is crucial, as it enables physicians to intervene earlier, optimize treatment, and improve outcomes for patients with these pulmonary conditions. Moreover, as more advanced echocardiographic techniques become available, our understanding of the role of RV strain in pulmonary diseases will continue to evolve, leading to better-targeted treatments and improved survival rates.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

None.

CONFLICTS OF INTEREST

We have no conflict of interest

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AUTHOR CONTRIBUTION

Conceptualization: NDR; Data Curation: NDR, YFV, NM; Formal Analysis: NDR, YFV, NM; Investigation: NDR, YFV, NM; Project Administration: NDR, YFV, NM; Resources: NDR, YFV, NM; Methodology: NDR, YFV, NM; Software: NDR, YFV, NM; Visualization: NDR, YFV, NM; Supervision: NDR, YFV, NM; Validation: NDR, YFV, NM; Writing – Original Draft Preparation: NDR, YFV, NM; Writing – Review & Editing: NDR, YFV, NM. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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