Right Ventricular Strain in Post-COVID Syndrome: Functional Recovery Does Not Always Mean Myocardial Recovery

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Among the various cardiovascular manifestations of post-COVID-19 syndrome, few remain as challenging as interpreting the persistent alterations in right ventricular mechanics. Although numerous studies have shown that the right ventricle is particularly susceptible to the overload imposed by the pulmonary circulation during the acute phase of infection, the clinical significance of the echocardiographic abnormalities that persist after recovery is still incompletely understood.[1]

Its high sensitivity to variations in pulmonary afterload, hypoxia, vascular inflammation, and microvascular thrombosis explains the vulnerability of the right ventricle to SARS-CoV-2. This fact justifies the growing interest in the right ventricular free-wall longitudinal strain (RVFWLS), currently recognized as the most sensitive echocardiographic method for detecting subclinical impairment of right ventricular function before changes appear in conventional parameters.[2,3]

The main merit of the present study does not lie merely in confirming the persistence of RVFWLS abnormalities after COVID-19 — a finding already described in previous investigations — but in shifting the discussion toward a conceptually more relevant question: does clinical recovery necessarily accompany the recovery of ventricular mechanics?[4,5]

This change in perspective represents, in my view, the study's principal scientific contribution. Rather than treating strain solely as a marker of ventricular dysfunction, the authors introduce the possibility that it may represent a biomarker of myocardial recovery trajectory. This concept significantly broadens its clinical interpretation, positioning strain as a potential biomarker of the biological recovery of the right ventricle, allowing clinicians to track remodeling processes that do not necessarily manifest in the patient's clinical course.

Although previous studies have demonstrated persistent impairment of RVFWLS after COVID-19, most of them focused on describing the presence of these abnormalities. The present work goes beyond this descriptive approach by exploring the biological meaning of the persistence of altered right ventricular strain during clinical follow-up, shifting the focus from simply identifying dysfunction to understanding myocardial recovery over time.[4,5]

The study's results reveal an apparent dissociation between clinical evolution and the evolution of right ventricular mechanics. Despite objective improvement in functional capacity and pulmonary diffusion over approximately one year of follow-up, RVFWLS remained mildly reduced and essentially stable, while conventional parameters of right ventricular function remained preserved.

At first glance, the absence of a robust association between strain and functional recovery could suggest limited clinical utility of RVFWLS. However, this interpretation overlooks a fundamental issue: mechanical biomarkers do not necessarily follow the same timeline as clinical recovery. On the contrary, the findings suggest that functional recovery and myocardial recovery represent physiologically distinct dimensions of the cardiovascular repair process. From this perspective, the persistence of subtle RVFWLS abnormalities ceases to represent a mere residual echocardiographic finding and instead reflects a potential marker of the biological trajectory of ventricular remodeling.

In this perspective, RVFWLS may represent a persistent mechanical signature of prior cardiopulmonary injury rather than an isolated marker of the patient's current clinical status.

This interpretation is biologically plausible. Myocardial deformation likely reflects not only momentary ventricular contractility but also the structural consequences of the injury sustained during the acute phase of the disease. Remodeling of the extracellular matrix, reorganization of myocardial fibers, persistence of microvascular abnormalities, and prolonged adaptation to changes in pulmonary afterload may sustain subtle alterations in ventricular mechanics even after partial recovery of pulmonary function and functional capacity.[1,5]

The study also reinforces a conceptual shift occurring in contemporary echocardiography. The debate is no longer about whether strain is superior to tricuspid annular plane systolic excursion (TAPSE) or factional area change (FAC) – a question largely answered by the literature – but rather about understanding which physiological dimensions each parameter represents.[2,3]

While TAPSE, FAC, and tricuspid annular systolic velocity reflect distinct components of ventricular function that depend on geometry and loading conditions, RVFWLS appears to capture earlier and more subtle changes in myocardial mechanics. Thus, strain should not be interpreted as a substitute for conventional parameters, but as a complementary component of a truly multiparametric assessment of right ventricular function, as emphasized by recent guidelines from the American Society of Echocardiography.[2,3]

Keywords

Heart Ventricles; Post-Acute COVID-19 Syndrome; Echocardiography; Global Longitudinal Strain

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In other words, ventricular mechanical function and functional performance appear to represent distinct dimensions of post-COVID recovery.

From this perspective, the article significantly expands the potential role of RVFWLS. Instead of using it solely as a diagnostic or prognostic marker, it opens the possibility of employing it as a tool to monitor ventricular remodeling over time. This view brings the assessment of the right ventricle closer to the contemporary concept of ventricular reserve and right ventricle–pulmonary circulation coupling, areas that will likely represent the next major advance in functional echocardiography.[2]

Naturally, some limitations must be considered. The absence of pre-infection echocardiograms makes it impossible to assert that all observed alterations resulted exclusively from COVID-19, especially in a population with a high prevalence of obesity, hypertension, and diabetes. Moreover, the relatively small number of participants reduces the statistical power to detect subtle associations, and the lack of cardiopulmonary exercise testing or invasive hemodynamic assessment limits more robust pathophysiological interpretations.[1,6]

Despite these limitations, the study offers an important conceptual contribution. It demonstrates that functional recovery may occur in parallel with the persistence of subtle alterations in right ventricular mechanics, reinforcing that the evaluation of these patients must remain integrated and multiparametric. More than answering whether the right ventricle improves after COVID-19, the work suggests that we may be assessing distinct physiological phenomena: on one hand, the patient's clinical recovery; on the other, the structural and mechanical recovery of the myocardium.

This distinction may extend beyond the context of COVID-19 and become applicable to other conditions characterized by acute injury to the right ventricle, such as pulmonary embolism, acute respiratory distress syndrome, pulmonary hypertension, and heart failure. In this sense, the present study contributes not only to the understanding of post-COVID syndrome but also to broadening our interpretation of the true clinical meaning of right ventricular free-wall longitudinal strain.[1,2]

The true advance of this study does not lie in demonstrating that RVFWLS remains altered after COVID-19. Its greatest merit is suggesting that ventricular mechanics follow their own timeline of recovery, partially independent of the patient's clinical evolution. This paradigm shift significantly expands the role of strain, which becomes interpreted not only as a marker of dysfunction but also as a potential biomarker of longitudinal myocardial remodeling.

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