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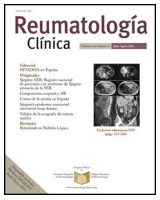

Letter to the Editor

Right ventriculoarterial coupling as a marker of subclinical myocardial damage in rheumatoid arthritis: Comments on the study by Miranda-Aquino et al.

Acoplamiento ventrículo-arterial derecho como marcador de daño miocárdico subclínico en la artritis reumatoide: Comentarios sobre el estudio de Miranda-Aquino et al.

To the Editor,

We read with great interest the recent article by Miranda-Aquino et al. entitled "Right ventriculoarterial coupling as a marker of subclinical myocardial damage in rheumatoid arthritis" published in your journal.[1] The authors report that patients with rheumatoid arthritis (RA) exhibited significantly reduced right ventriculo-arterial coupling (RVAC, calculated as right ventricular free wall longitudinal strain [RVFWLS]/pulmonary artery systolic pressure [PASP]) compared with matched healthy controls, and that RA diagnosis was independently associated with abnormal RVAC.

We congratulate the authors on addressing the under-investigated area of right ventricular (RV)–pulmonary arterial coupling in RA, a domain of increasing importance given the recognised elevated cardiovascular (CV) risk in RA populations.[2–4] Their findings contribute meaningfully to the emerging field of cardio-rheumatology by suggesting that RVAC, a non-invasive echocardiographic metric, may unmask subclinical RV myocardial damage prior to overt dysfunction or pulmonary hypertension.

Nevertheless, we would like to highlight several points that merit further discussion and may guide future research.

Integration into routine cardiac screening algorithms in RA

While the demonstration of altered RVAC in RA is compelling, the question remains how this parameter might be integrated into routine CV screening in rheumatology practice. Current EULAR recommendations emphasise traditional CV risk factor management (hypertension, dyslipidaemia, smoking cessation) and left-ventricular diastolic function evaluation but do not specify RVAC measurement.[5] Given the study's cross-sectional design, it remains uncertain whether abnormal RVAC predicts clinically relevant outcomes (e.g., right heart failure or CV events) in RA. Therefore, establishing the independent and incremental prognostic value of RVAC over established risk markers is a critical next step before its widespread clinical adoption. Longitudinal studies correlating RVAC trajectories with clinical outcomes would provide this missing link.

Influence of RA treatment, disease activity and inflammatory burden

A notable strength of the study was the homogeneous inclusion of RA patients without major comorbidities, reducing confounding. However, the prevalence of abnormal RVAC was not stratified by key rheumatologic parameters: (i) disease activity indices (e.g., DAS28, SDAI), (ii) DMARD or biologic exposure, and (iii) cumulative glucocorticoid dose. Because both systemic inflammation and therapeutic modulation affect CV risk,[6–8] it would be valuable to adjust for these variables. For instance, long-term methotrexate or biologic therapy may ameliorate strain abnormalities, whereas persistent inflammation may worsen RV remodelling. Incorporating such adjustments could refine interpretation of whether the RA diagnosis itself, or rather the cumulative inflammatory burden and its modulation by treatment, is the primary driver of RVAC abnormalities.

Standardisation of measurement and cut-off values

The authors used the RVFWLS/PASP ratio to estimate RVAC, an elegant speckle-tracking strain-based approach. However, widespread clinical adoption may be challenged by inter-vendor variability, the inherent load-dependence of strain measurements, and the need for RA-specific normative data. Prior studies in non-RA cohorts demonstrated that TAPSE/PASP, S'/PASP, and RVFAC/RVSP ratios correlate well with invasive Ees/Ea coupling and predict outcomes in heart failure and pulmonary hypertension.[3,9] Defining reproducible cut-off values and assessing intra-observer variability across echocardiographic platforms will be essential to ensure methodological consistency.

Clinical implications and potential therapeutic ramifications

If validated, RVAC could enable earlier cardioprotective intervention in RA, including intensified anti-inflammatory therapy or CV risk management. As a hypothetical framework for future research, the authors might consider proposing an algorithm where RA patients undergo baseline echocardiography incorporating RVAC; those with abnormal coupling are prioritised for cardio-rheumatology consultation and serial monitoring. Furthermore, longitudinal RVAC assessment could serve as a sensitive surrogate endpoint in studies evaluating the cardioprotective effects of disease-modifying therapy.[6,8,9]

Conclusion

The study by Miranda-Aquino et al. significantly broadens our understanding of subclinical right-sided myocardial involvement

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in RA. Further research should focus on (i) establishing prognostic value, (ii) clarifying the influence of inflammation and therapy, and (iii) standardising echocardiographic methodology. Until then, heightened vigilance for right-heart dysfunction and close rheumatology–cardiology collaboration remain essential for comprehensive CV care in RA.[2,5,8]

Ethical approval

Not applicable.

Declaration of generative AI and AI-assisted technologies in the writing process

The authors declare that no generative AI or AI-assisted technologies were used in the preparation of this manuscript. All content was fully developed, written, and reviewed by the authors.

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Conflict of interest

The author declares no conflicts of interest relevant to this article.

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