Short Editorial

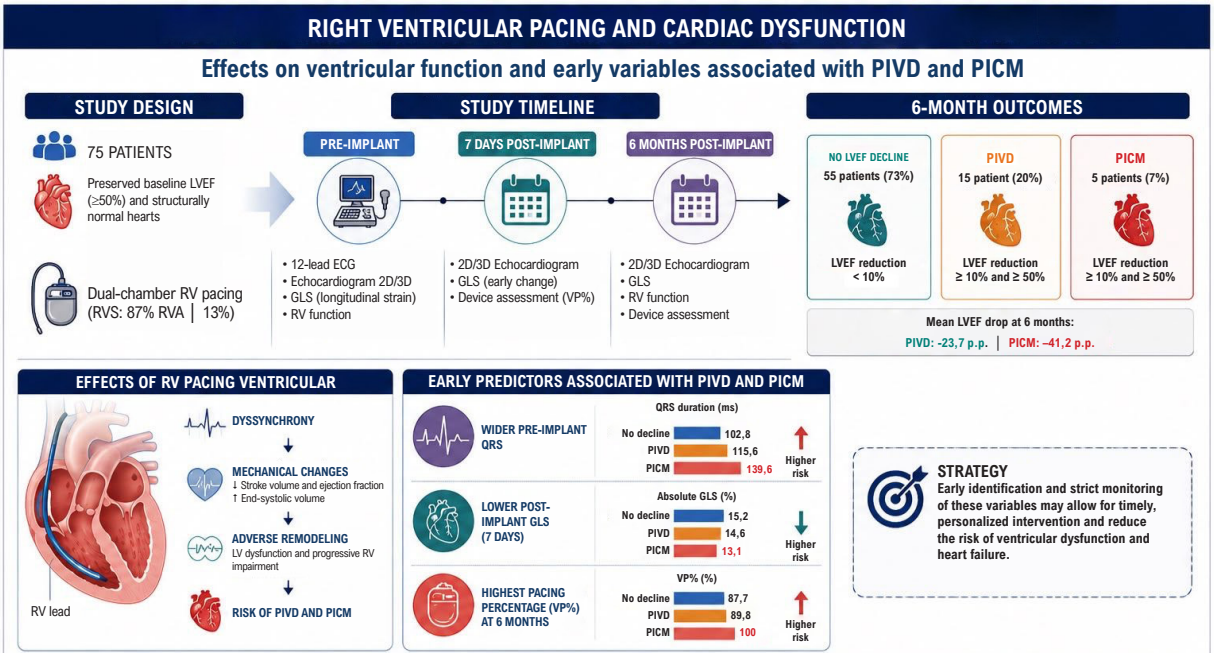
Beyond Ejection Fraction: In Pacemaker-Induced Cardiomyopathy, are We Closer to Identifying At-Risk Patients Early?

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Short Editorial related to the article: Effects of Pacemakers on Ventricular Functions and Early Variables Associated with Induced Cardiomyopathy

Central Illustration: Beyond Ejection Fraction: In Pacemaker-Induced Cardiomyopathy, are We Closer to Identifying At-Risk Patients Early?



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The natural history of symptomatic bradyarrhythmias was greatly impacted from the 1950s onwards with the implementation of cardiac pacemakers, initiating the era of Artificial Cardiac Stimulation.[1] Dual-chamber pacemakers are routinely used in advanced atrioventricular blocks in structurally normal hearts.[2] Right ventricular pacing, despite its effectiveness in correcting conduction disturbances, has deleterious effects on ventricular mechanics.[1] It promotes ventricular dyssynchrony, triggering a reduction in systolic function and, in a portion of patients, the development of pacemaker-induced cardiomyopathy (PICM).[3] In this context, in addition to ejection fraction, the identification of risk predictors for ventricular dysfunction (VID) and PICM allows for better clinical decision-making and a reduction in the impact of heart failure in patients dependent on right ventricular pacing.

This exploratory study presents global longitudinal strain (GLS) as an early marker of myocardial impairment. Although left ventricular ejection fraction (LVEF) remains the most widely used metric in clinical practice, its reduction is known to often represent a relatively late stage of functional injury.[3] In contrast, GLS allows the identification of subclinical changes

Keywords

Heart Failure; Artificial Pacemaker; Cardiac Resynchronization Therapy Devices.

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in longitudinal contractility before significant changes in LVEF occur. The study shows that GLS reductions are early, already observable as early as seven days after implantation in patients who subsequently developed IVP or PICM. The possibility of identifying vulnerable patients as early as the first week after implantation is particularly attractive.

Another relevant aspect was the association between longer baseline QRS complex duration and subsequent occurrence of ventricular dysfunction. Patients who developed PICM presented a significantly wider native QRS complex before implantation. This finding suggests that previous alterations in intraventricular conduction may represent a favorable substrate for the development of a greater degree of dyssynchrony after artificial stimulation.[3,4]

Similarly, the high percentage of ventricular pacing emerged as another marker associated with adverse outcomes.[3,4] The group that developed PICM showed virtually complete dependence on ventricular pacing during follow-up. This result is aligned with previous evidence demonstrating a direct relationship between cumulative right ventricular pacing load and progressive deterioration of left ventricular function. From a clinical perspective, these data reinforce the importance of careful device programming and the adoption of strategies aimed at minimizing unnecessary ventricular pacing whenever possible.

Naturally, the results must be interpreted in light of certain limitations. This is a single-center study with a relatively small sample size and short follow-up period. The small number of PICM cases limited robust multivariate analyses and prevents definitive conclusions about causality. Despite these limitations, the clinical message of the study is clear. In patients undergoing right ventricular pacing, the combination of a wider baseline QRS complex, early reduction in GLS, and a high pacing percentage identifies a phenotype at higher risk for ventricular remodeling. The systematic incorporation of these variables into the assessment may allow for more intensive monitoring and early selection of patients eligible for more physiological pacing strategies, such as conduction system pacing or, in selected situations, cardiac resynchronization therapy.

As we move toward an increasingly personalized approach to heart failure, studies like this reinforce the need to shift from a strategy based solely on the late identification of LVEF decline to models that value early markers of myocardial vulnerability.

References

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