

Isolated right ventricular non-compaction in a post-MI elderly patient: a diagnostic blind spot in geriatric cardiology

Andrea Elia^{1,✉}, Gian Luca Iannuzzi²

1. Alzheimer's Center at Temple (ACT), Lewis Katz School of Medicine, Temple University, Philadelphia, Pennsylvania, USA; 2. Local Health Authority, Service of Cardiology, 82100 Benevento, Italy

✉ Correspondence to: andrea.elia@temple.edu

<https://doi.org/10.26599/1671-5411.2026.05.001>

Isolated Ventricular Non-Compaction (IVNC) is characterized by prominent trabeculations and deep intertrabecular recesses in the ventricular myocardium,^[1] believed to result from arrested myocardial compaction during embryogenesis.^[2,3] While most literature focuses on left ventricular non-compaction (LVNC),^[4,5] isolated involvement of the right ventricle (IRVNC)^[6-9] is underreported. Diagnostic criteria remain largely derived from left ventricular definitions (e.g., Jenni, *et al.*^[10]), and right heart functional implications are poorly characterized. In older adults, especially those with coronary artery disease (CAD), recognition of IRVNC may be delayed, misattributed to more common etiologies of right heart failure.^[11,12]

We present a rare case of IRVNC in a 65-year-old male following an inferior non-ST-elevation myocardial infarction (NSTEMI), which revealed the diagnostic and therapeutic challenges this entity poses in elderly populations. Following successful coronary angioplasty, the patient enrolled in a structured cardiac rehabilitation program. Physical exam was unremarkable except for a mild mitral end-systolic murmur; resting oxygen saturation about 89%; heart rate about 55 beats/min. ECG showed sinus bradycardia and evidence of recent inferior MI. 24-hour Holter revealed complex ventricular arrhythmias (Figure 1). The echocardiographic evaluation demonstrated normal cardiac dimensions and preserved systolic-diastolic function, with a left ventricular ejection fraction (LVEF) of 60% (biplane Simpson), and 55% by the TEI index-derived method. Left ventricular filling pressures were normal (E/E' : 7), and these findings were corroborated by Obokata's formula ($E/E' \times S'$: 1.2).^[13] Mild mitral regurgitation was noted. High-resol-

ution echocardiography confirmed isolated RV non-compaction (Figure 2A-B), with normal right ventricular systolic function as evidenced by Tricuspid Annular Plane Systolic Excursion (TAPSE: 21 mm), right ventricular fractional area change (RVFAC: 37%), and S' wave at Tissue Doppler Imaging (TDI: 13). However, elevated right ventricular filling pressures were detected (E/E' : 9) (Figure 2C). Tricuspid valvular and right ventricular TDI patterns ruled out right ventricular diastolic dysfunction. The estimated pulmonary artery pressure (ePAP) was at the upper limit of normal (systolic ePAP: 30 mmHg; mean ePAP: 22 mmHg). No ventricular involvement was left evident.

The clinical course was marked by episodes of decompensation during rehabilitation, despite preserved systolic indices and guideline-directed medical therapy. Diuretic intensification led to symptomatic improvement, and at discharge, the patient's NYHA class improved from III to II with substantial reduction in perceived exertional dyspnea (Borg RPE: from 8 to 3). The arrhythmic burden on Holter was deemed more likely attributable to prior infarction rather than IVNC, and implantable cardioverter-defibrillator (ICD) placement was deferred.

This case serves to challenge prevailing assumptions about IVNC as predominantly a left ventricular disease and highlights the potential for right ventricular non-compaction to masquerade as unexplained right heart failure, particularly in geriatric patients with concomitant coronary disease. In our patient, CAD and IRVNC co-existed, and while the former was treated percutaneously, the latter remained a persistent and underappreciated contributor to clinical symptoms.

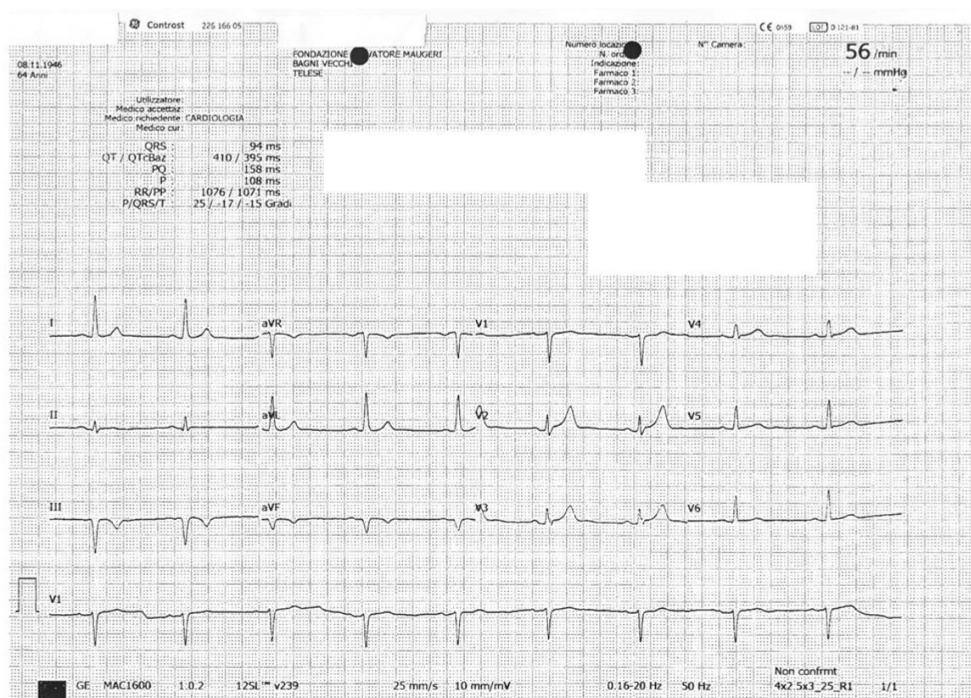


Figure 1 RVINC is not associated with sinus beat impairment. The patient's electrocardiogram on recovery showed sinus bradycardia and recent inferior myocardial infarction. RVINC: right ventricular isolated non-compaction.

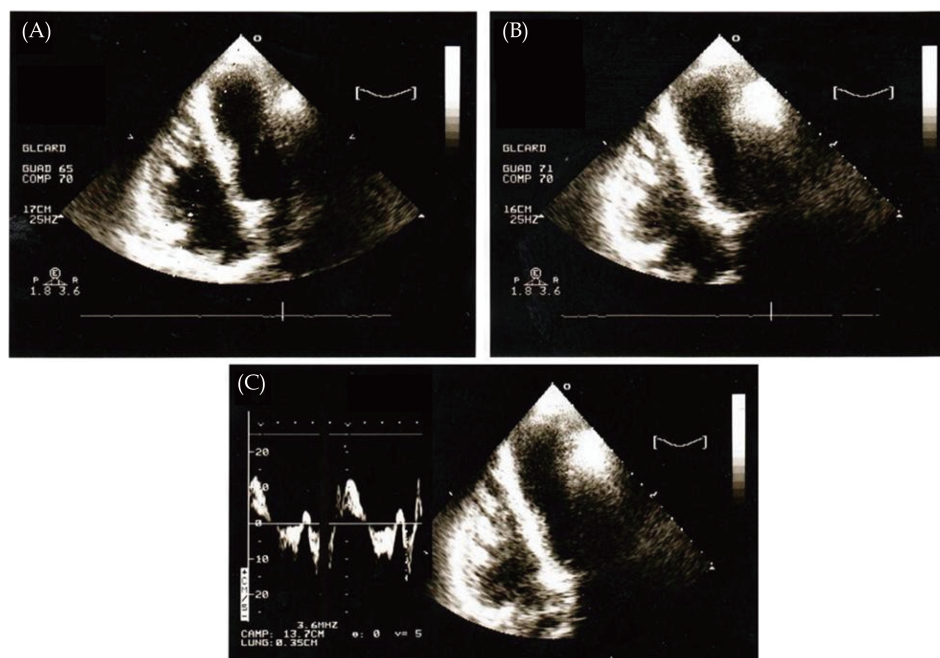


Figure 2 Echocardiographic assessment of RVINC. (A): Four-chamber views of the echocardiogram highlighting right ventricular hypertrabeculation; (B): Magnified view of the right ventricular hypertrabeculation, showing the “spongy myocardium” with the red arrow indicating the affected region; (C): TDI of the right ventricle, depicting normal systolic function and elevated right ventricular filling pressures. RVINC: right ventricular isolated non-compaction; TDI: Tissue Doppler Imaging.

Two critical considerations emerge: (1) diagnostic vigilance is essential: In elderly patients with preserved left heart function and unexplained right heart failure symptoms, IRVNC should be a differential consideration, especially when traditional etiologies (e.g., pulmonary hypertension, valvular disease) are excluded. (2) Refined imaging protocols are urgently needed: Current diagnostic criteria for RVNC are neither standardized nor widely accepted. The absence of CMR or 3D echocardiography in our case is a limitation yet also reflects real-world challenges in routine practice. Echocardiographers must remain alert to trabecular anomalies even in the absence of established RVNC guidelines.

While the etiopathogenesis of IVNC remains debated, whether genetic, mechanical, or both, it is increasingly clear that ventricular hypertrabeculation is not always benign, particularly when associated with clinical decompensation, as in this case. Furthermore, the lack of prior echocardiographic data in this patient underscores the importance of baseline cardiac imaging in elderly individuals with risk factors, even in the absence of overt symptoms.

In conclusion, we urge clinicians and researchers to reconsider the traditional left-centric lens through which IVNC is viewed. Right ventricular forms of the disease deserve equal attention, especially given their potential to present subtly yet evolve toward significant morbidity if unrecognized. Future studies must establish validated RVNC-specific diagnostic criteria and investigate tailored therapeutic strategies to guide clinicians facing this elusive diagnosis.

DISCLOSURE

Data Availability Statement

The corresponding author will share the data underlying this article upon reasonable request.

Ethics Statement

The patient provided their written informed consent to participate in this study. Written informed consent was obtained from the individual for the publication of any potentially identifiable images or data included in this article.

Funding

This work was supported by American Heart Association

Postdoctoral fellowship 24POST1240115 awarded to AE.

Acknowledgments

The authors disclose any financial and personal relationships with other people or organizations that could inappropriately influence their work. The authors declare no financial support. The authors wish to thank Domenica Rosa Grasso with gratitude for her technical support.

REFERENCES

- [1] Angelini A, Melacini P, Barbero F, *et al.* Evolutionary persistence of spongy myocardium in humans. *Circulation* 1999; 99: 2475.
- [2] Kayvanpour E, Sedaghat-Hamedani F, Gi WT, *et al.* Clinical and genetic insights into non-compaction: a meta-analysis and systematic review on 7598 individuals. *Clin Res Cardiol* 2019; 108: 1297–1308.
- [3] van Waning JJ, *et al.* Systematic Review of Genotype-Phenotype Correlations in Noncompaction Cardiomyopathy. *J Am Heart Assoc* 2019; 8: e012993.
- [4] Arbustini E, Favalli V, Narula N, *et al.* Left Ventricular Noncompaction: A Distinct Genetic Cardiomyopathy? *J Am Coll Cardiol* 2016; 68: 949–966.
- [5] Pittorru R, De Lazzari M, Migliore F, *et al.* Left Ventricular Non-Compaction: Evolving Concepts. *J Clin Med* 2024; 13: 5674.
- [6] Gardner MM, Cohen MS. Clinical findings in right ventricular noncompaction in hypoplastic left heart syndrome. *Congenit Heart Dis* 2017; 12: 783–786.
- [7] Yu W, Thomas MA, Mills L, *et al.* Prenatal Diagnosis of Isolated Right Ventricular Non-Compaction Cardiomyopathy with an MYH7 Likely Pathogenic Variant. *Fetal Pediatr Pathol* 2023; 42: 464–471.
- [8] Stämpfli SF, Gotschy A, Kiarostami P, *et al.* Right ventricular involvement in left ventricular non-compaction cardiomyopathy. *Cardiol J* 2022; 29: 454–462.
- [9] Huang PP, Tang YX, Huang XS. A Rare Case of Isolated Right Ventricular Non-compaction With the Novel TTN Mutation. *Front Cardiovasc Med* 2022; 9: 845973.
- [10] Jenni R, Oechslin E, Schneider J, *et al.* Echocardiographic and pathoanatomical characteristics of isolated noncompaction: a step towards classification as a distinct cardiomyopathy. *Heart* 2001; 86: 666–671.
- [11] Adamo M, Chioncel O, Pagnesi M, *et al.* Epidemiology, pathophysiology, diagnosis and management of chronic right-sided heart failure and tricuspid regurgitation. A clinical consensus statement of the Heart Failure Association (HFA) and the European Association of Percutaneous Cardiovascular Interventions (EAPCI) of the ESC. *Eur J Heart Fail* 2024; 26: 18–33.
- [12] Goldstein JA, Lerakis S, Moreno PR. Right ventricular



myocardial infarction — a tale of two ventricles: JACC Focus Seminar 1/5. *J Am Coll Cardiol* 2024; 83: 1779–1798.

[13] Mornoş C, Petrescu L, Ionac A, *et al*. The prognostic value of a new tissue Doppler parameter in patients with heart failure. *Int J Cardiovasc Imaging* 2014; 30: 47–55.

Please cite this article as: Elia1 A, Iannuzzi GL. Isolated right ventricular non-compaction in a post-MI elderly patient: a diagnostic blind spot in geriatric cardiology. *J Geriatr Cardiol* 2026; 23(5): 322–325. DOI: 10.26599/1671-5411.2026.05.001

