The Cyprus Journal of Cardiovascular Medicine

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A message from the President of the Society

Dear colleagues, dear friends

It is with great satisfaction that I address, on behalf of the Board of Directors of the Cyprus Society of Cardiology, the anniversary volume of 'The Cyprus Journal of Cardiovascular Medicine', the official journal of the Cyprus Society of Cardiology. This volume is published within the frameworks of the 30th Conference 'Cardiology Today', the biennial international conference of our Society.

Reaching such a milestone is a testament to the Society's enduring commitment to advancing cardiovascular science, continuous education and clinical practice.

It is particularly encouraging to see that this special volume includes abstracts submitted by cardiology residents and medical students. Providing a platform for early-career cardiologists and potential doctors to present their work is invaluable for the development of the next generation of cardiologists and for foster ing a culture of academic curiosity and innovation.

The inclusion of these contributions not only highlights the vitality of emerging talent in the field but also strengthens the academic community by encouraging young investigators to engage in scientific dialogue and research dissemination.

I wish the journal continued success in promoting quality research and supporting the growth of future leaders in cardiology. To the editorial board, I extent my sincere appreciation and I thank them for their contribution.

With my warmest regards and wishes for a fruitful and productive meeting,

Kyriakos Yiangou
President,
Cyprus Society of Cardiology

EDITORIAL

European Harmonization of Cardiology Training: A Strategic Opportunity for Cyprus

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Abstract
Cardiovascular disease (CVD) remains the leading cause of mortality across Europe, demanding consistently high standards of specialist education. Despite structured initiatives by the European Society of Cardiology (ESC), including the Core Curriculum for the Cardiologist and the European Examination in Core Cardiology (EECC), substantial heterogeneity persists in cardiology training among European countries. Differences in procedural exposure, competency validation, subspecialty accreditation, and research integration challenge the objective of uniform excellence in cardiovascular care. For smaller member states such as Cyprus, harmonization represents not only alignment with European standards but a strategic opportunity to elevate training quality, enhance international credibility, and strengthen patient outcomes. This editorial explores the clinical, educational, and policy dimensions of unified cardiology education across Europe.

The European Cardiovascular Burden and the Educational Imperative
Cardiovascular disease accounts for nearly four million deaths annually across Europe and approximately 45% of total mortality [1]. Advances in interventional cardiology, structural heart disease therapies, electrophysiology, multimodality imaging, heart failure management, and preventive cardiology have transformed the specialty into one of the most technologically demanding domains in medicine.

This increasing complexity imposes a professional responsibility: cardiology training must be rigorous, measurable, and comparable across borders. In a European Union characterized by professional mobility, shared clinical guidelines, and cross-border collaboration, fragmented educational standards are increasingly misaligned with clinical reality.

Persistent Heterogeneity in Training Structures
Although cardiology specialization typically follows internal medicine training, European systems vary significantly in the duration of training (4–6 years in most countries), in procedural volume requirements for coronary angiography, PCI, and device implantation, in access to sub-

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specialty rotations (electrophysiology, structural interventions, advanced imaging), in research obligations and in methods of competency assessment.

The ESC Core Curriculum (2020 update) provides a comprehensive framework defining knowledge, skills, and professional attitudes required for independent practice [2]. However, its implementation depends on national regulatory authorities and remains inconsistent.

Directive 2005/36/EC ensures recognition of professional qualifications within the European Union [3]. Yet it emphasizes duration and certification rather than demonstrable competency. Consequently, equivalence of title does not necessarily imply equivalence of training experience.

From Time-Based Training to Competency-Based Education

Modern medical education increasingly favours competency-based medical education (CBME) over time-based apprenticeship [4]. Entrustable Professional Activities (EPAs) allow structured evaluation of readiness for independent clinical responsibility.

In cardiology, harmonized competencies should include independent management of acute coronary syndromes, safe performance and interpretation of coronary angiographies, cardiac device implantation and complication management, multimodality imaging interpretation and comprehensive cardiovascular prevention strategies.

Without common competency validation, procedural autonomy may be inconsistently granted, introducing variability in clinical confidence and patient safety.

The Strategic Role of the EECC

The EECC was established to provide a pan-European knowledge benchmark aligned with the ESC Core Curriculum [5]. It represents a critical step toward cognitive standardization.

However, examination alone cannot assess procedural proficiency, clinical judgment under stress, multidisciplinary collaboration, or communication skills. A unified European framework should therefore integrate knowledge certification (EECC), workplace-based competency assessment, simulation-based validation and accreditation of training centers.

Such integration would ensure that certification reflects both knowledge and performance.

Technological Acceleration and Training Demands

Recent years have seen rapid expansion in transcatheter valve therapies, in complex PCI techniques, in pacing systems, in advanced heart failure treatment including devices and in artificial intelligence in imaging.

Structured exposure to such technologies is uneven across Europe. Smaller healthcare systems may lack sufficient procedural volumes to guarantee consistent experience. Without structured cross-border fellowship pathways, disparities may widen.

A harmonized European training model could facilitate rotational subspecialty exposure while preserving national training bases.

Preventive Cardiology as a Core Competency

The 2021 ESC Guidelines on cardiovascular disease prevention emphasize comprehensive risk factor control and lifestyle modification [6]. Preventive cardiology must occupy a central position in training programs.

Structured exposure to lipid management clinics, hypertension services, diabetes-cardiology and kidney interfaces, and lifestyle intervention programs should be mandatory components of

harmonized curricula.

For Cyprus, where demographic shifts and cardiovascular risk factors remain significant, strengthening preventive cardiology education directly influences national health outcomes.

Workforce Mobility and Professional Equity

Europe faces uneven distribution of cardiologists and subspecialists (7). Harmonization enhances mobility while maintaining quality assurance.

For Cyprus, European alignment offers strategic advantages, such as access to advanced subspecialty fellowships, strengthened academic collaboration, participation in multinational registries and research networks and enhanced professional credibility.

Barriers to Harmonization

Challenges include, national regulatory sovereignty, economic disparities in healthcare infrastructure, language diversity and institutional resistance to reform.

These obstacles require gradual implementation, collaborative dialogue, and respect for national frameworks while maintaining European minimum standards.

A Practical Roadmap

A structured pathway toward harmonization may include, adoption of the ESC Core Curriculum as a national minimum benchmark, recognition of the EECC as a knowledge certification endpoint, accreditation of cardiology training centers, digital cross-border logbooks documenting procedural exposure, simulation-based competency validation, structured European fellowship exchanges and periodic recertification aligned with continuing professional development.

Such measures enhance quality without compromising national autonomy.

Conclusion

The harmonization of cardiology education across Europe is not merely an academic aspiration; it represents a structural evolution in response to the growing complexity of cardiovascular medicine. Shared standards in training, competency assessment, and certification are increasingly necessary to safeguard patient outcomes, facilitate professional mobility, and sustain scientific excellence.

For Cyprus, European harmonization may offer the possibility to consolidate training quality, enhance international credibility, attract academic collaboration, and ensure that Cypriot cardiologists operate within a transparent and measurable European benchmark of excellence. In a smaller healthcare system, structured integration into a unified European framework can mitigate limitations in subspecialty exposure while preserving national autonomy in organization and delivery.

The tools are already available: the ESC Core Curriculum, the European Examination in Core Cardiology, evolving competency-based educational models, and established European scientific networks. What is required now is deliberate institutional engagement and strategic vision.

If embraced thoughtfully, European harmonization of cardiology training can serve not as an external imposition, but as a catalyst for strengthening cardiovascular care in Cyprus—positioning the country not at the periphery, but as an active and credible participant in shaping the future of European cardiology.

Key Messages

- Cardiovascular disease remains the leading cause of mortality in Europe, requiring uniformly high standards in specialist cardiology training.
- Despite the ESC Core Curriculum and the

EECC, significant heterogeneity persists across European training systems.
- Competency-based harmonization would enhance patient safety, professional mobility, and academic integration.
- For Cyprus, alignment with a unified European training framework represents a strategic opportunity to strengthen national standards, international credibility, and cardiovascular outcomes.

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CARDIOLOGY UPDATE 2026

30-Day Outcomes of Transfemoral Valve-in-Valve Transcatheter Aortic Valve Implantation from Nicosia GH TAVI registry

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Introduction

Valve-in-valve (ViV) transcatheter aortic valve implantation (TAVI) is an established treatment option for degenerated surgical bioprostheses. However data on hemodynamic performance by valve type remain limited.

Methods

Aiming to evaluate 30-day clinical and hemodynamic outcomes of transfemoral ViV TAVI, including comparison of post-procedural gradients between self-expanding and balloon-expandable valves, we retrospectively analyzed 30 consecutive patients undergoing transfemoral ViV TAVI (including 5 TAVI-in-TAVI cases) at our center, since 2015 (all after 2020; 3.3% of 919 total TAVIs). Valve selection was at operator discretion (56.7% self-expanding Evolut, 43.3% balloon-expandable Sapien 3). Baseline characteristics, pre- and post-procedural aortic valve (AV) gradients and 30-day adverse events were assessed per VARC-3 criteria where applicable. Independent samples t-tests compared post-procedural gradients between valve types.

Results

Mean age was 77.5 ± 6.8 years, 70% were male. Comorbitities included heart failure (30%), atrial fibrillation (46.7%), diabetes (40%) and prior CABG/PCI (13.3%/43.3%). Patients' risk profiles were EuroSCORE II high-risk 50%, STS high-risk 33.3%. Self-expanding valves were deployed in smaller annuli (pre-AV diameter 18.8 ± 2.1 mm vs. 22.3 ± 2.9 mm, $p<0.001$). Post-procedural gradients were low overall but significantly lower with self-expanding vs. balloon-expandable valves (peak: 12.5 ± 5.2 vs. 20.1 ± 8.2 mmHg, $p=0.02$; mean: 6.4 ± 2.6 vs. 11.2 ± 4.5 mmHg, $p=0.009$). At 30 days, all-cause mortality was 0%, cardiac death 0%, disabling stroke 3.3% (n=1), minor vascular complications 10%, major vascular 0%, new pacemaker 0%, and MACE 3.3%.

Conclusion

Transfemoral ViV TAVI yielded excellent 30-day safety with zero mortality and low MACE. Self-expanding valves achieved superior hemodynamic performance (lower gradients) despite smaller baseline annuli, supporting their preferential use in ViV procedures where feasible.

CITATION: Karmioti G., Eteocleous N., Zittis I., Eftychiou Ch., Myrianthopoulos K., Mitsis A. 30-Day Outcomes of Transfemoral Valve-in-Valve Transcatheter Aortic Valve Implantation from Nicosia GH TAVI registry, CyJCardMed 2026, Volume 4, Issue 1, 7-9

PATIENTS' CHARACTERISTICS	
Characteristic	***301 patients***
Age	77.5 +/- 6.8 (55-88)
Male sex	(21) 70%
Heart failure	(9) 30%
CABG	(4) 13.3%
Previous PCI	(13) 43.3%
Permanent Pacemaker	(8) 26.7%
Atrial Fibrillation	(14) 46.7%
Previous Stroke	(3) 10%
Peripheral Vascular Disease	(5) 26.7%
Pulmonary Hypertension	(7) 23.3%
Chronic Lung Disease	(6) 20%
Chronic Kidney Disease	(9) 30%
Diabetes Mellitus	(12) 40%
History of Chest Radiation	(1) 3.3%
Porcelain Aorta	(4) 13.3%
Euroscore II category	
· low risk	(5) 16.7%
· Intermediate risk	(10) 33.3%
· High risk	(15) 50%
STS category	
· low risk	(8) 26.7%
· Intermediate risk	(12) 40%
· High risk	(10) 33.3%

Table 1

30 DAY RESULTS OF TF VIV TAVI	
Cardiac Death	(0) 0%
All cause Death	(0) 0%
Disabling Stroke	(1) 3.3%
Minor Vascular Complications	(3) 10.7%
Major Vascular Complications	(0) 0%
MACE	(1) 3.3%
Pacemaker	(0) 0%

Table 3

ViV TAVI cases	*Sapien 3 (13, 43.3%)*	*Evolut (17, 56.7%)*	*P value*
AV area	431+/-106mm3	282+/-66.5 mm3	<0.001
AV diameter	22.3+/-2.9mm	18.8+/-2.1mm	<0.001
AV perimeter	73.6+/-8.3mm	59.5+/-7.2mm	<0.001
Pre peak AV gr	43.5+/-29.4 mmHg	67+/-27 mmHg	0.125
Pre mean AV gr	26.8+/-20.4 mmHg	38.6+/- 16.6mmHg	0.177
Post peak AV gr	20.1+/-8.2 mmHg	12.5+/-5.2 mmHg	0.02
Post mean AV gr	11.2+/-4.5mmHg	6.4+/-2.6mmHg	0.009

Table 2

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Ajmaline challenge for Brugada Syndrome: the experience of the Nicosia General Hospital in the era of the new Cyprus Healthcare System

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Introduction

Ajmaline challenge testing is widely used to unmask the diagnostic Type 1 electrocardiographic pattern in patients with suspected Brugada syndrome[1]. By inhibiting cardiac sodium channels, ajmaline accentuates ST-segment elevation, facilitating diagnosis and informing clinical risk stratification[2]. The test has demonstrated good diagnostic performance when performed under controlled conditions with appropriate monitoring[3]. We report our 5-year experience from the Nicosia General Hospital and its dedicated multidisciplinary team.

Keywords: Ajmaline challenge, Brugada syndrome, risk stratification, genetics

Methods

Retrospective registry analysis conducted by three independent reviewers, integrating data from multiple patient records.

Results

Between August 2020 and August 2025, 51 ajmaline challenges were performed (35 males, 16 females). Median patient age was 39 years (IQR: 30–48 years); 92% were of Cypriot origin. The positivity rate was 35%, with male predominance (12 males vs. 6 females). Common referral indications included suspicious symptoms (presyncope/syncope), strong family history of sudden cardiac death or Brugada syndrome, and suggestive electrocardiographic findings (non-specific J-point elevation or Type 2 pattern). No procedural complications occurred. Among patients with positive tests, three underwent implantable loop recorder insertion for syncope evaluation, and two received an implantable cardioverter-defibrillator due to combined syncope and family history. Genetic testing identified one likely pathogenic variant in SCN10A, while other identified variants in CTNNA3, DMD, BAG3, FLNC, CACNA1D, and ALPK3 genes were not typically consistent with Brugada syndrome.

CITATION: Demetriades V., Fouseki A., Ioannides M., Katsikas Ch., Christophides Th., Ajmaline challenge for Brugada Syndrome: the experience of the Nicosia General Hospital in the era of the new Cyprus Healthcare System CyJCardMed 2026, Volume 4, Issue 1, 10-11

No pathogenic SCN5A variants have been detected so far.

Conclusions

Ajmaline challenge proved safe and diagnostically valuable in our cohort. This first formal report in Cyprus supports the establishment of a national inherited arrhythmia registry to enhance systematic data collection and risk stratification.

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Myocardial Bridging in a Symptomatic Medical Student

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Abstract

A 25-year-old male medical student presented with effort-induced chest pain and ECG changes mimicking acute coronary syndrome. Investigations revealed a myocardial bridge in the mid-left anterior descending (LAD) artery as the cause, with no obstructive coronary disease. Beta-blocker therapy provided symptomatic relief. This case highlights myocardial bridging as a key differential in young patients with ischemia-like symptoms and underscores the role of medical optimization.

Introduction

Myocardial bridging (MB) is a congenital anomaly in which an epicardial coronary artery segment, most commonly the mid-LAD, courses intramurally through the myocardium, resulting in systolic compression. Autopsy and CT angiography studies report a prevalence of 5-25%, yet most cases remain asymptomatic. Symptomatic MB can manifest as angina, arrhythmias, acute coronary syndromes, or sudden death, particularly in young, low-risk individuals presenting as myocardial infarction with non-obstructive coronary arteries (MINOCA). We report a case of effort-induced angina due to MB in a medical student, emphasizing its diagnostic challenges and management.

Case Presentation

A 25-year-old male medical student presented to the emergency department with a 3-week history of substernal chest pain triggered by physical exertion and exam-related stress. Symptoms worsened with increased caffeine and energy drink consumption but were absent at rest. He denied nausea, vomiting, reflux, dyspnea at rest, or recreational drug use. Family history was notable for premature coronary artery disease in his father (myocardial infarction at age 45) and paternal grandfather. Vital signs on arrival: heart rate 100 bpm (regular rhythm), blood pressure 155/95 mmHg, respiratory rate 18/min, SpO2 99% on room air, temperature 36.7°C. Physical examination was unremarkable, with no cardiac murmurs, rubs, or gallops. Laboratory results, including high-sensitivity troponin T, B-type natriuretic peptide (BNP), D-dimer, complete blood count, and metabolic panel, were normal. Chest X-ray showed no acute abnormalities.

CITATION: J Van Heerden P., F Travers W., Myocardial Bridging in a Symptomatic Medical Student CyJCardMed 2026, Volume 4, Issue 1, 12-15

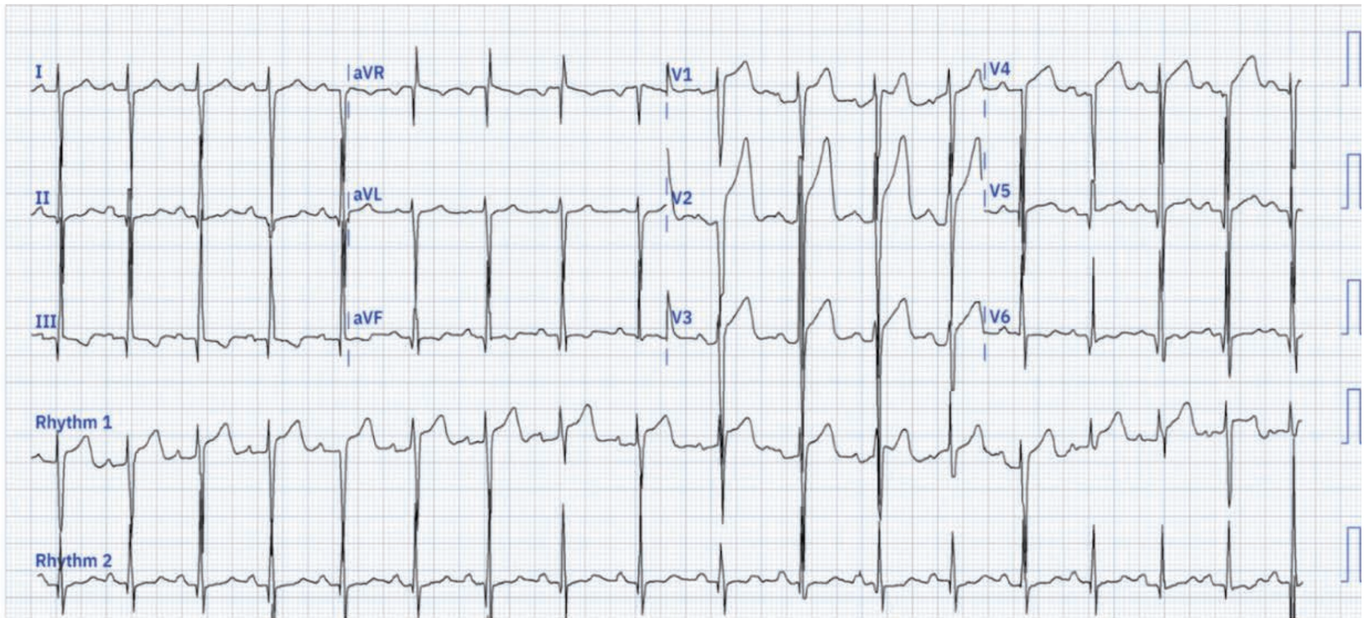

Figure 1: *12-lead electrocardiogram demonstrating sinus tachycardia (HR 100 bpm), right axis deviation (+120°), concave ST-segment elevation (2-3 mm) in leads V1-V5, and reciprocal ST depression (1 mm) in inferior leads II, III, aVF. These findings mimic acute anterior ST-elevation myocardial infarction but resolved with beta-blocker therapy, consistent with demand ischemia from myocardial bridging.*

Investigations

12-lead ECG revealed sinus tachycardia with right axis deviation (+120°), concave ST-segment elevation (2-3 mm) in V1-V5, and reciprocal ST depression (1 mm) in II, III, aVF *(Figure 1)*. Differentials included acute anterior ST-elevation myocardial infarction (STEMI), hypertrophic cardiomyopathy, pulmonary embolism, or acute pericarditis/myocarditis.

Transthoracic echocardiography demonstrated normal biventricular size and systolic function (ejection fraction 62%), no wall motion abnormalities, no pericardial effusion, and normal right ventricular systolic pressure.

Coronary computed tomography angiography (CCTA) identified a significant myocardial bridge in the mid-LAD with >50% systolic narrowing but no atherosclerosis or plaque. Invasive coronary angiography con-

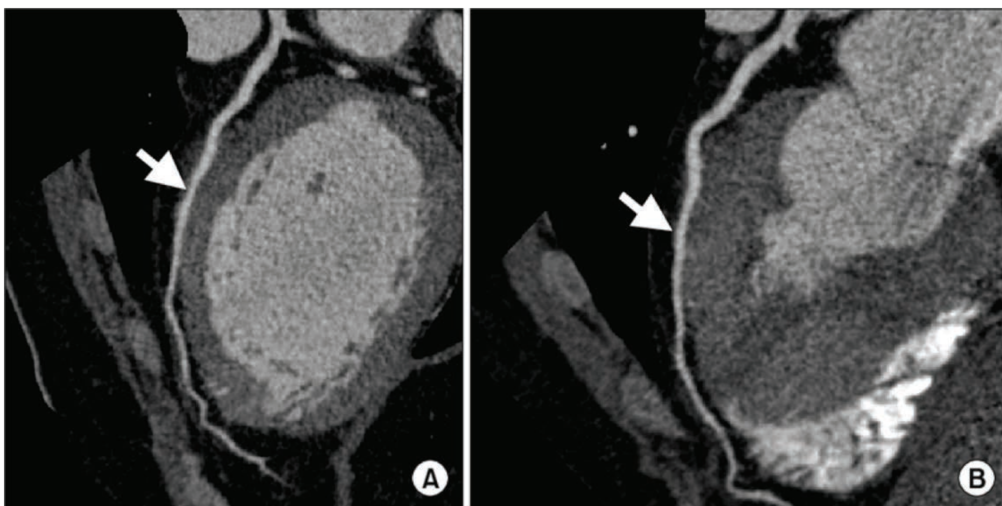

Figure 2: *Coronary computed tomography angiography (CCTA) in a systolic phase (75% R-R interval) showing a significant myocardial bridge in the mid-left anterior descending (LAD) artery with >50% systolic narrowing (arrow). No atherosclerosis or plaque is evident in proximal or distal segments. Diastolic phase images confirmed normal vessel caliber.*

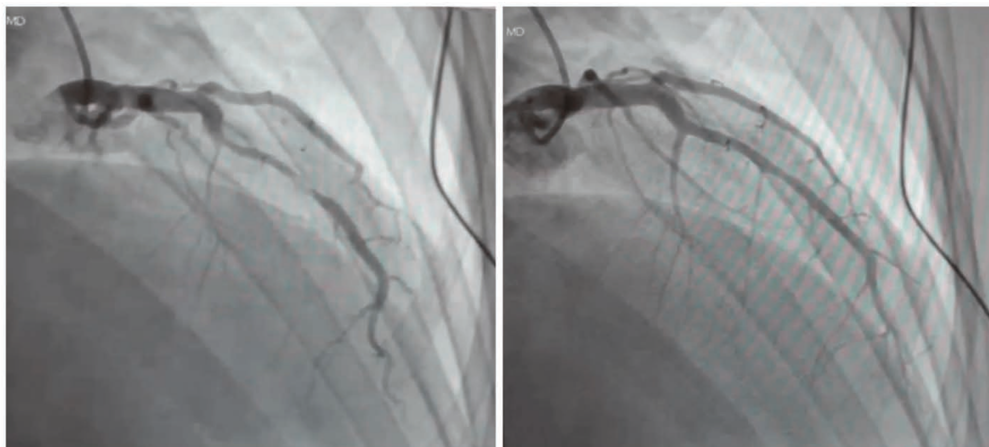

Figure 3: *Invasive coronary angiography of the left anterior descending (LAD) artery in systole, demonstrating characteristic "milking effect" with systolic compression of the mid-LAD segment.No fixed stenoses or atherosclerotic disease.*

firmed these findings, showing "milking" of the bridged segment during systole without fixed stenoses *(Figure 3)*

Differential Diagnosis and Management

Initial differentials—STEMI, hypertrophic cardiomyopathy, pulmonary embolism, and myocarditis—were excluded by normal serial troponins, echocardiography, and D-dimer. MB was diagnosed as the etiology of demand ischemia, consistent with MINOCA guidelines.

The patient was started on metoprolol succinate 50 mg daily, titrated to 100 mg for heart rate control <70 bpm at rest. He was advised to avoid nitrates (which exacerbate systolic compression) and limit caffeine/energy drinks. At 3-month follow-up, he was asymptomatic with normalized ECG and blood pressure 130/80 mmHg on therapy.

Discussion

Symptomatic MB, though rare clinically despite high anatomic prevalence, induces ischemia via systolic compression, delayed diastolic relaxation, and endothelial shear stress. Mid-LAD bridges confer higher risk, particularly during exertion or tachycardia, as seen here. ECG changes mimicking STEMI are well-described but often transient. Beta-blockers are first-line, reducing contractility and heart rate to minimize compression and prolong diastolic perfusion; non-dihydropyridine calcium channel blockers are alternatives. Refractory cases may warrant surgical unroofing, stenting, or bypass, though evidence is limited to case series.

This case illustrates MB as a treatable cause of angina in low-risk youth, urging prompt angiography in atypical presentations.

Conclusion

Early recognition of MB via CCTA or angiography enables targeted beta-blocker therapy and excellent prognosis. Clinicians should consider MB in young adults with exertional chest pain and ECG abnormalities, even with normal troponins.

Keywords: Hypertension, Blood Pressure Control, European Society of Hypertension (ESH) Guidelines 2024, Combination Therapy, Treatment Adherence.

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Reversible LV Dysfunction and Functional Mitral Regurgitation in End-Stage Kidney Disease: Complete Reverse Remodeling after Kidney Transplantation

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Introduction

Patients with end-stage kidney disease (ESKD) frequently develop left ventricular (LV) systolic dysfunction and functional mitral regurgitation (MR), both of which are associated with adverse cardiovascular outcomes[1,2]. Although advanced heart failure may complicate transplant candidacy, emerging evidence suggests that renal transplantation can significantly improve cardiac structure and function, including reversal of LV dysfunction1. Functional MR in this setting is primarily driven by LV remodelling and altered ventricular geometry[3].

Method

This single-patient case report utilized serial multimodality cardiac imaging, including transthoracic and transesophageal echocardiography, stress cardiac magnetic resonance, and exercise stress echocardiography to evaluate LV size, systolic function, MR severity, ischemia, fibrosis, and contractile reserve. Guideline-directed heart failure therapy was implemented, and renal graft function was monitored longitudinally.

Results

A 47-year-old man with ESKD on hemodialysis, previously with normal cardiac function, developed new LV dilation (6.0 cm), reduced LVEF (~40%), and moderate functional MR prior to kidney transplantation. Early after transplantation, he presented with acute pulmonary edema. Transesophageal echocardiography demonstrated severely dilated LV, LVEF 35–40%, and severe secondary MR (Carpentier IIIB/I). Stress CMR confirmed severe LV dilation with moderately impaired systolic function (LVEF 44%), without inducible ischemia or myocardial fibrosis, consistent with non-ischemic dilated cardiomyopathy. Following transplantation and optimized medical therapy, progressive reverse remodeling occurred. At 6-month follow-up post-transplantation, LV size had normalized, LVEF improved to 60%, and mitral regurgitation had

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completely resolved at rest and during exercise.

Conclusions:

Renal transplantation can lead to substantial improvement in cardiac structure and function, even in patients with severe LV systolic dysfunction at baseline. Restoration of renal function may promote reverse remodeling through correction of chronic volume overload, normalization of preload and afterload conditions, reduction of arteriovenous fistula–related high-flow hemodynamic stress, and reversal of uremic myocardial toxicity[1,2]. Functional MR, largely driven by ventricular remodelling, may significantly improve as left ventricular geometry and contractile function recover[3].These findings support transplantation as a viable therapeutic strategy in carefully selected ESKD patients with concomitant heart failure.

Key words: End-stage kidney disease, LV systolic dysfunction, Functional mitral regurgitation, Reverse remodeling, Kidney transplantation

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Early Mechanical Circulatory Support with Impella CP in Fulminant Myocarditis Leading to Recovery of Left Ventricular Function – Case series

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Background

Early recognition and timely intervention are essential in fulminant myocarditis, a rapidly progressive inflammatory cardiomyopathy that often leads to cardiogenic shock and multiorgan failure1. Despite its severe initial presentation, fulminant myocarditis may have a favourable long-term prognosis when promptly recognized and treated[1,2]. Mechanical circulatory support (MCS), including venoarterial extracorporeal membrane oxygenation, represents a crucial therapeutic strategy to stabilize patients, maintain end-organ perfusion, and facilitate myocardial recovery in refractory cases[3].

Cases

We present a case series of three young adults who presented with acute decompensated heart failure and cardiogenic shock following viral infections. Echocardiography revealed severely reduced left ventricular ejection fraction (LVEF) in all cases. An Impella CP device was implanted in each patient to provide left ventricular unloading and hemodynamic support. Over a short duration of support, all patients demonstrated progressive improvement in LVEF and systemic perfusion, enabling safe device removal. All patients were discharged on guideline-directed medical therapy with recovery of ventricular function.

Discussion

This case series highlights the role of Impella as an important adjunct in the management of fulminant myocarditis complicated by cardiogenic shock. By reducing left ventricular workload and optimizing perfusion, Impella facilitates myocardial rest and recovery, potentially reducing the need for durable mechanical support or transplantation. Close hemodynamic monitoring and a tailored weaning strategy were key to the favourable outcome observed.

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Conclusion

In fulminant myocarditis with cardiogenic shock, the use of Impella can provide effective hemodynamic stabilization and support myocardial recovery, reinforcing its value as an important option within a multidisciplinary management approach.

Keywords: fulminant myocarditis, Impella CP, cardiogenic shock, mechanical circulatory support

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Prevalence of High Blood Pressure in Patients with Previously Diagnosed Hypertension

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One of the leading causes of cardiovascular disease and early mortality worldwide is hypertension. Many people with documented hypertension continue to report values over recommended targets during routine clinical assessment, despite the existence of clear management guidelines, including well-defined blood pressure thresholds. Improving patient outcomes and guaranteeing compliance with national standards depend on an understanding of the degree of uncontrolled blood pressure in this population.

This audit aimed to identify the proportion of patients with an established diagnosis of hypertension who were not meeting age-specific BP targets based on NICE guideline NG136 (2025). At Evangelismos Private Hospital, a retrospective cross-sectional review of adult patients with established hypertension who were at least 18 years old was conducted. The provided dataset was used to extract patient age and clinic blood pressure readings. BP greater than 140/90 mmHg for those under 80 years of age and greater than 150/90 mmHg for those over 80 was the target control. The percentage of people reaching or surpassing suggested levels was calculated using descriptive analysis, and results were compared to NICE quality benchmarks to assess present performance. The audit reveals areas where management or follow-up procedures might need to be strengthened and offers information about local BP control levels.

Introduction

Hypertension is the leading cause of cardiovascular and premature deaths worldwide. Although significant advances have been made in antihypertensive therapy, the worldwide burden of hypertension is continuously rising. In 2010, an estimated 1.39 billion adults which is almost a third of the global population, were affected by hypertension, with low and middle-income countries having a much higher incidence rate than high-income nations.

Lifestyle factors such as dietary salt intake, obesity, alcohol consumption, lack of physical activity, and broader socioeconomic issues all contribute to regional differences in prevalence and results. Furthermore , despite the fact that hypertension is widely recognised and treatable,

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global rates of awareness, treatment, and effective blood pressure control remain low, showcasing ongoing challenges in detection, long-term management, and cardiovascular risk reduction (Mills et al., 2020).

The NICE NG136 guidelines aid the diagnosis and management of hypertension in the United Kingdom. According to these criteria, a clinic blood pressure of 140/90 mmHg or greater is indicative of hypertension, but confirmation should be obtained from continuous home blood pressure monitoring, where the diagnostic threshold is slightly lower at 135/85 mmHg. Once a diagnosis has been verified, therapy goals are categorized by age. Adults under the age of 80 are considered managed if their clinic blood pressure remains below 140/90 mmHg, while those above the age of 80 should aim for less than 150/90 mmHg.These standards are based on research that shows that keeping blood pressure within these ranges significantly reduces the risk of myocardial infarction, stroke, chronic renal disease, and heart failure, making them an important quality target in normal clinical care (NICE, 2023).

Despite the simple nature of these standards, a significant minority of patients with hypertension do not meet their recommended blood pressure levels. National data show that many persons remain uncontrolled even when treatment and follow-up alternatives are available. Recent population level study has revealed ongoing gaps in the hypertension control cascade, including medication adherence, treatment intensification, and continuity of care. Uncontrolled hypertension is highly connected with long-term cardiovascular complications and is an alarming indicator of unmet clinical need. The proportion of patients with diagnosed hypertension who remain above target thresholds provides useful information on the efficacy of current clinical practice and the delivery of guideline-based care. A local audit enables comparison with national standards and the identification of short-comings with potential for better administration and follow-up (Richardson et al., 2024).

Standards

S1: Patients <80 years should have a clinic BP <140/90 mmHg (Target: 70–80%).

S2: Patients ≥80 years should have a clinic BP <150/90 mmHg (Target: 70–80%).

S3: All hypertensive patients should have a documented clinic BP reading (Target: 100%).

Methodology

This retrospective clinical audit took place at Evangelismos Private Hospital in Paphos, Cyprus. The goal was to evaluate blood pressure (BP) control in adults with high blood pressure. We included all patients aged 18 years and older who had a confirmed diagnosis of hypertension and a documented clinic BP reading. Patients without a recorded BP measurement or without a confirmed hypertension diagnosis were excluded. We extracted data from an anonymized clinical dataset.

We based our audit standards on NICE NG136 (2023). This guideline sets the target clinic BP at less than 140/90 mmHg for adults under 80 years and less than 150/90 mmHg for those aged 80 years and older. The expected control rate is between 70 and 80%. Another standard required 100% documentation of clinic BP.

The extracted variables included age, systolic and diastolic BP, diabetes status, and the class of antihypertensive medication. We divided patients into two groups: those under 80 and those 80 and older. We used descriptive statistics to

calculate the proportion of patients meeting BP targets, the prevalence of diabetes, and patterns of medication use. We compared the results directly with NICE standards. All data were handled anonymously, and we did not need ethical approval since this work met the criteria for clinical audit and quality improvement.

Results

TABLE 1: POPULATION CHARACTERISTICS

Variable	*Result*
Number of patients (n)	58
Mean age (yrs/std)	74 +/- 10.211
Age group <80 (n, %)	33 (56.90)
Age group >=80 (n, %)	25 (43.1)
Sex distribution	M:38 F: 20
Average systolic/ diastolic BP <80 (mmHg, SD)	144.15/82.1
Average systolic/ diastolic BP >= 80 (mmHg, SD)	143.48/74.16

TABLE 2: OUTCOMES FOR POPULATION UNDER INVESTIGATION

Age Group	Target BP	At Target (n, %)	Above Target (n,%)	Presence of DM (n,%)	ACE/ARB Therapy	CCB	Thiazide diuretics	B-Blockers	Meets standard BP target?
<80	<140/90 mmHg	19 (57.57)	14 (42.43)	10 (30.30)	18 (54.54)	12 (36.36)	7 (21.21)	14 (42.42)	No
>=80	<150/90 mmHg	13 (52)	12 (48)	11 (44)	22 (88)	8 (32)	14 (56)	18 (72)	No
Total		32 (55.17)	26 (44.83)	21 (36)	40 (68.96)	20 (34.48)	21 (36.21)	32 (55.17)	No

TABLE 3: BP CONTROL AND DIABETES

	Bp controlled (n, %)	Bp uncontrolled (n, %)	Total (n)
Diabetic	9 (42.9%)	12 (57.1%)	21
Non-diabetic	19 (51.4%)	18 (48.6%)	37

***Note:** *Calculation: Relative Risk (RR) = 0.429/0.514 = 0.83; Odds ratio (OR) = (9x18)/(12x19) = 162/228 = 0.71. Chi-square (X2) ≈ 0.41, p-value ≈ 0.52* *Note: Calculation: Relative Risk (RR) = 0.429/0.514 = 0.83; Odds ratio (OR) = (9x18)/(12x19) = 162/228 = 0.71. Chi-square (X2) ≈ 0.41, p-value ≈ 0.52*

Figure 1. BP control vs Standard

Figure 12. BP Control and Diabetes Prevalence

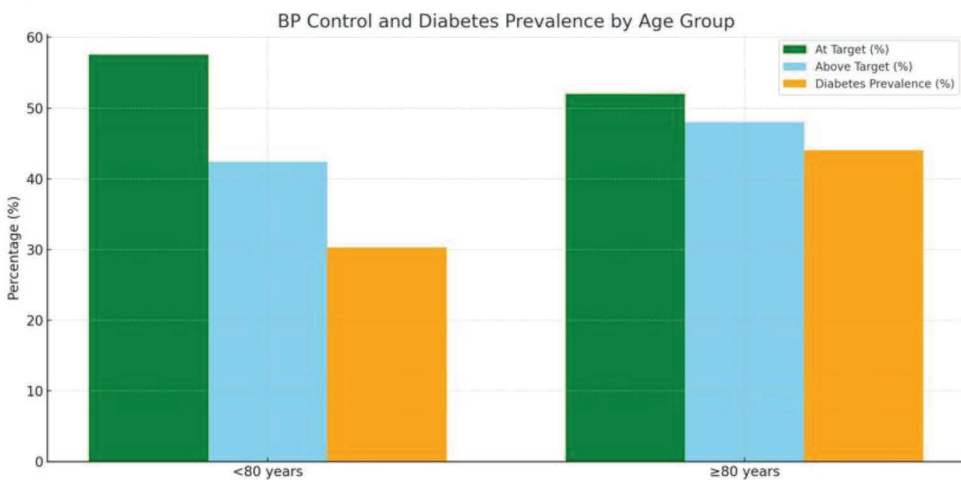


Figure 3. Medication class use by age group

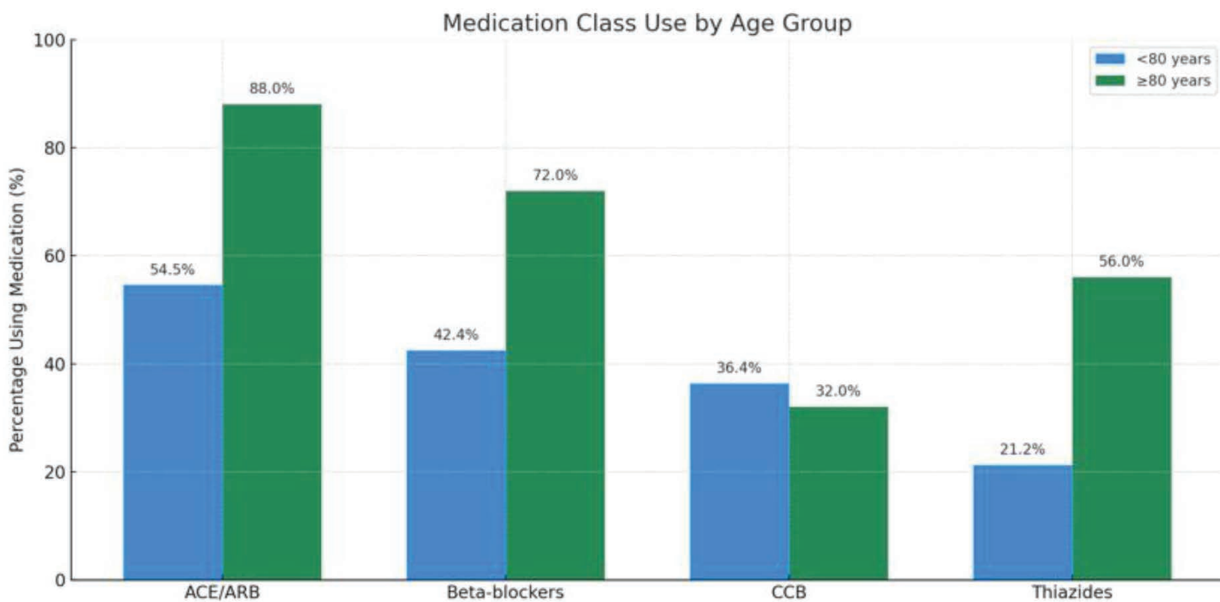
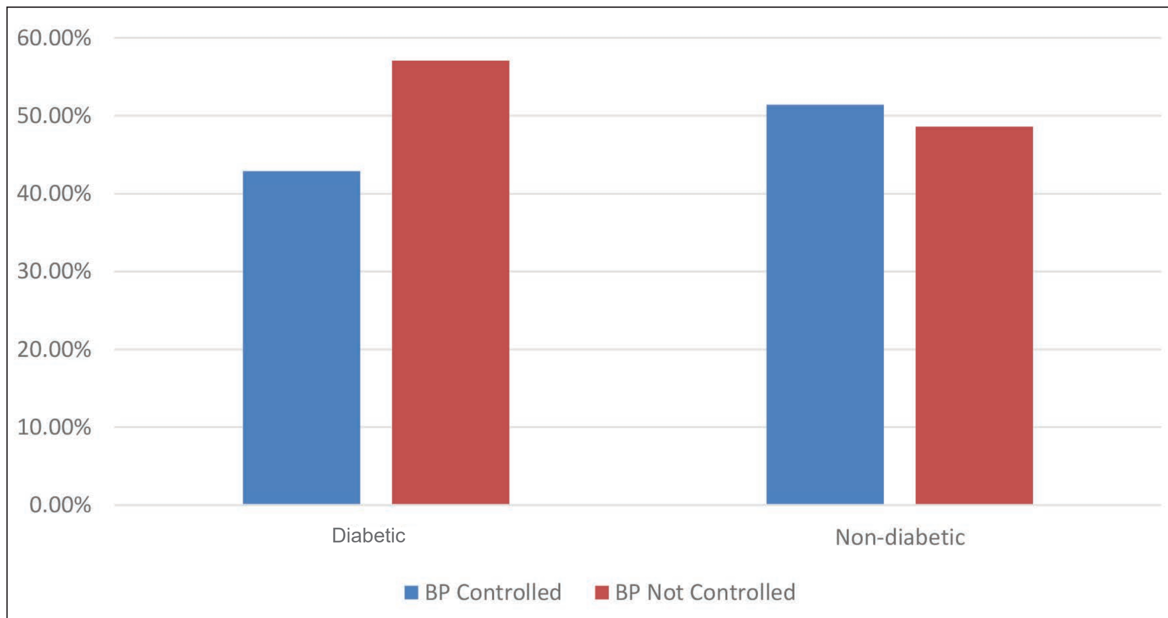


Figure 4. BP control and diabetes



The study included 58 patients, of which 33 (56.9%) were under the age of 80 and 25 (43.1%) were 80 years or older. The mean age was 74 years, with a male-to-female ratio of 38 to 20 respectively. The average clinic blood pressure between the age groups were similar. The <80 group had an average reading of 144/82 mmHg, while for the >= 80 group it was 143/74 mmHg. The audit criteria indicates that the target blood pressure differs between the two groups. For patients aged <80, the target reading was <140/90 mmHg. Of the 33 patients in that group 19 met the criteria (57.6%), while 14 (42.4%) failed to. In the >=80 age group, whose target was <150/90 mmHg, 13 out of 25 patients (52%) achieved the target reading, the remaining 12 patients (48%) didn't meet the criteria. Combining both groups 32 of the 58 (55.2%) were at target, whilst 26 (44.8%) were above the target. Neither group met the target threshold of 70-80%.

All patients irrespective of age group had a recorded clinic blood pressure reading, this complies with S3 (100%).

Figure 2 includes information on the prevalence of diabetes mellitus (DM) amongst the age groups. 10 individuals (30.3%) in the <80 group had DM. In comparison, 11 individuals (44%) in the >=80 cohort had DM. pooled together, an overall of 21 from the 58 patients presented with the disease.

Figure 3 demonstrates medication patterns per age group. In the <80 population 18 patients (54.5%) were on ACE inhibitors or ARBs, compared with the 22 patients (88%) in the >=80 group. Calcium channel blockers (CCB) were prescribed to 12 (36.3%) and 8 (32%) of the patients aged <80 and >=80 respectively. Thiazide diuretics were prescribed to 7 patients (21.2%) in the younger group and 14 patients (56%) in the older. Beta-blockers were used.

Table 3 and **figure 4** demonstrate the differences between blood pressure management between patients with and without diabetes. The diabetic cohort demonstrated poorer blood pressure controller (42.9%) than the non-diabetics (51.4%). An 8.5% better BP control is seen in

non-diabetics. Diabetic patients in this cohort were 17% less likely to achieve blood pressure control than non-diabetics. Although the difference was not statistically significant (X2 =0.41, p = 0.52) by 14 patients (42.4%) aged under 80 and 18 patients (72%) aged 80 or above. When ranking the medications prescribed in order of most to least used the list goes, ACE/ARB therapy, (40 patients, 69%), followed by Beta-blockers (32 patients, 55%), thiazide diuretics (21 patients, 36%) and calcium channel blockers (20 patients, 34%).

Overall, a lower-than-target BP control in both age groups is seen when compared to the audit standards, and differences are seen in medication class utilization between age groups. The results also showcase a substantial prevalence of DM in both age groups but slightly higher in the older population.

Discussion

This audit examined how well patients with a confirmed diagnosis of hypertension at Evangelismos Private Hospital achieved age-appropriate blood pressure (BP) control, as defined by the NICE guideline NG136. This guideline suggests maintaining clinic BP below 140/90 mmHg for adults under 80 years old and below 150/90 mmHg for adults aged 80 years and older (NICE 2023). These targets match international evidence that shows significant reductions in cardiovascular events when BP stays within these ranges (SPRINT Research Group 2015). Overall, this audit found that BP control in this group is below recommended levels, indicating increased long-term cardiovascular risk for the Cypriot population.

Among the 58 patients reviewed, only 57.6% of adults under 80 and 52% of those 80 or older met the target BP levels, which is well below the NICE audit goal of 70-80%. This finding corresponds with global analyses that highlight persistent suboptimal BP control in various populations, even with improvements in diagnosis and treatment options (Zhou et al. 2021). Comparisons with international guidelines reveal similar challenges. The 2018 European Society of Cardiology/European Society of Hypertension (ESC/ESH) guidelines urge more aggressive control for high-risk groups. However, real-world data from Europe still show inadequate BP management, especially among older adults (Williams et al. 2018).

Given Cyprus's ageing population, notably in areas like Paphos, the low control rates found in this audit signal a critical need for better local cardiovascular prevention strategies.

The prevalence of diabetes was higher in those aged 80 and older (44% compared to 30.3% in those under 80), likely affecting BP control negatively. The combination of hypertension and diabetes is known to significantly increase cardiovascular risk, due to factors like endothelial dysfunction, autonomic neuropathy, and microvascular disease (American Diabetes Association 2024). This aligns with data from the Mediterranean region, which shows rising diabetes rates linked to lifestyle issues such as diet, decreased physical activity, and obesity. The persistent coexistence of diabetes and hypertension in Cyprus reflects findings from the European Heart Network, which states that cardiovascular disease is a leading cause of death in Mediterranean regions (European Heart Network 2023). Thus, the high diabetes burden in this group emphasizes the need for tailored treatment strategies that consider multiple health conditions.

Blood pressure control was also lower among the diabetic cohort (42.9% vs 51.4%). Diabetic patients were 17% (RR = 0.83) less likely

to achieve blood pressure control than their counterpart. this is consistent with existing literature, and it showcases that it is more difficult to manage mtension in that group. Though this result was not statistically significant, most likely due to the lower sample size of the audit (Paulsen et al., 2013).

Patterns of medication use showed notable age-related differences. ACE inhibitors or ARBs were the most prescribed medications, especially for older adults (88%). This was followed by beta-blockers, thiazide diuretics, and calcium channel blockers. These prescribing trends partly align with ESC/ESH and ACC/AHA recommendations, both of which endorse RAAS inhibition as a first-line treatment for many patients, particularly those with diabetes or kidney issues (Whelton et al. 2018). However, despite relatively high medication usage, BP control remained inadequate. This suggests potential problems such as not intensifying treatment, concerns about polypharmacy, underuse of combination therapy, or challenges with adherence. These are common issues noted in European hypertension studies (Zhou et al. 2021).

Strengths of this audit include a clearly defined standard, complete BP documentation that meets S3, and the inclusion of data on comorbidities and medications. Limitations include the modest sample size, the single-centre setting, and the lack of information on adherence, treatment duration, and lifestyle factors. Moreover, the cross-sectional design does not allow for assessment of long-term BP stability.

Based on these findings, targeted recommendations include improving pathways for medication optimization, introducing earlier combination therapy, giving priority to patients aged 80 and older and those with diabetes for better follow-ups, enhancing lifestyle interventions based on Mediterranean guidance, and improving documentation of comorbidities and adherence. Implementing structured BP review clinics or digital follow-up tools may also help improve control. A re-audit within 6-12 months is suggested to assess progress after these interventions.

Conclusion

The audit revealed that although full compliance with documentation standards were met (S3), important gaps were found between current blood pressure outcomes of patients at Evangelismos Private Hospital with previously diagnosed hypertension and the target standard under the guidelines. This indicates presence of non-compliance and/or that current management strategies are not achieving optimal outcomes in terms of cardiovascular protection. Although it is clearly seen that antihypertensive therapy is prescribed to most patients, further refinement of treatment pathways may be needed to ensure maximal cardiovascular protection for the patients.

The audit also emphasizes how significant it is to take action sooner for people with additional risk factors such as older aged individuals and/or those with comorbid diseases. Suggestions for improvement of long-term patient outcomes includes, Enhancement of follow-up systems, counselling or education for issues such as lifestyle choices, improving care continuity, the integration of patient specific structured medication reviews and embedding evidence based cardiovascular prevention strategies.

Going forward, the implementation of targeted quality improvement measures and re-auditing within the next year will be essential for progress assessment. By addressing identified gaps, the medical team may work towards a more consistent adherence to target guideline standards and improvement of patient care.

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Severe Elevation of LDL-C Despite Combination Therapy: A Case Report Supporting Guideline-Directed PCSK9 Inhibition

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Background

Achieving and maintaining LDL-C targets in very high-risk patients remains a major challenge in secondary prevention. Current guidelines from the European Society of Cardiology recommend LDL-C <55 mg/dL and ≥50% reduction from baseline in patients with established atherosclerotic cardiovascular disease (ASCVD). Failure to maintain target levels despite maximally tolerated therapy necessitates treatment intensification.

Case Summary

We present a 71-year-old male with extensive ASCVD, including prior myocardial infarction, coronary angioplasty with stent implantation (2020), heart failure, hypertension, and type 2 diabetes mellitus with complications. The patient was receiving combination lipid-lowering therapy with rosuvastatin 20 mg and ezetimibe 10 mg, in addition to optimal secondary prevention treatment (aspirin, beta-blocker, antihypertensive therapy, and SGLT2 inhibitor).

Serial lipid monitoring demonstrated marked LDL-C increase:

December 2025: LDL-C 160 mg/dL

February 2026: LDL-C 248 mg/dL

LDL-C rose from 160 mg/dL to 248 mg/dL within 2 months, while triglycerides remained within normal limits. This represents a dramatic and progressive loss of lipid control despite ongoing combination therapy.

Given his very high cardiovascular risk profile and LDL-C levels >4-fold above the recommended target, the patient fulfils clear criteria for PCSK9 inhibitor initiation.

Discussion

This case illustrates the use of guidelines regarding LDL-C targets and real-world lipid control in secondary prevention. Persistent or rebound severe hypercholesterolemia despite statin–ezetimibe therapy should prompt early consideration of PCSK9 inhibition. Large out-

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come trials such as FOURIER and ODYSSEY OUTCOMES have demonstrated significant cardiovascular risk reduction with PCSK9 inhibitors in high-risk populations.

Conclusion

In very high-risk ASCVD patients, substantial LDL-C rebound despite maximally tolerated therapy represents a clear indication for PCSK9 inhibitor escalation. Early recognition and intervention are essential to mitigate recurrent cardiovascular risk.

Keywords: Atherosclerosis, LDL, Secondary prevention, PCSK9 inhibitors, Hypercholesterolemia

Introduction

Atherosclerotic cardiovascular disease (ASCVD) remains a leading cause of morbidity and mortality worldwide, with patients who have established hyperlipidaemia at particularly high risk of recurrent cardiovascular events. Effective lipid management, specifically reduction of low-density lipoprotein cholesterol (LDL-C), is a cornerstone of secondary prevention. Current European Society of Cardiology (ESC) guidelines recommend stringent LDL-C targets of less than 55 mg/dL and at least a 50% reduction from baseline in very high-risk patients [1].

Despite the availability of high-intensity statins and combination therapy with ezetimibe, a significant proportion of patients fail to achieve or maintain these targets in real-world clinical practice. This may be due to factors such as biological variability in response, disease progression, or limitations in therapy effectiveness. In such cases, guideline-directed management emphasizes treatment intensification.

Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors represent a major advancement in lipid-lowering therapy. By increasing LDL-receptor recycling, these agents produce substantial reductions in circulating LDL-C levels and have demonstrated cardiovascular benefit in large outcome trials. PCSK9 have an apolipoprotein B100 carrier protein that binds to LDL receptors on hepatocytes. Cellular uptake of the complex results in lysosomal destruction of LDL, resulting in decreased clearance of LDL in the bloodstream. PCSK9 inhibitors are monoclonal antibodies that bind to serum PCSK9 and subsequently prevent the destruction of LDL receptors *(Figure 1)*.[2]

This case report illustrates the clinical importance of recognizing severe LDL-C rebound and the need for timely escalation to PCSK9-targeted therapy.

Case Presentation

A 71-year-old male with a history of myocardial infarction, prior percutaneous coronary intervention (2020), heart failure, hypertension, and type 2 diabetes mellitus presented for routine follow-up. He was receiving guideline-directed medical therapy, including rosuvastatin 20 mg daily and ezetimibe 10 mg daily. Additional medications included aspirin, empagliflozin, bisoprolol, amlodipine, and folic acid.

The patient was asymptomatic, with no reported chest pain, dyspnoea, or functional decline. He denied smoking and alcohol use, and there was no known family history of premature cardiovascular disease. Data regarding body mass index and recent lifestyle changes were not available.

Over a 2-month period, laboratory results demonstrated a rapid deterioration in lipid control. On 11 December 2025, total cholesterol was 243 mg/dL and LDL-C 160 mg/dL; by 9 February 2026, total cholesterol had increased to 324 mg/dL and LDL-C to 248 mg/dL.

The magnitude and rapidity of LDL-C elevation were disproportionate to expected biological variability. The patient reported adherence to prescribed lipid-lowering therapy; however, objective adherence assessment was not available. Evaluation for secondary causes of dyslipidaemia revealed normal thyroid function, renal function, electrolytes, and glycaemic parameters.

This represents a marked loss of lipid control, with LDL-C rising to levels substantially exceeding recommended targets for very high-risk patients. Given the significant LDL-C elevation despite maximally tolerated statin–ezetimibe therapy and the absence of identifiable secondary causes, escalation to PCSK9 inhibitor therapy was pursued, and approval is currently pending.

Discussion

This case highlights a key challenge in secondary prevention: failure to maintain LDL-C targets despite guideline-directed combination therapy. While statins and ezetimibe form the foundation of lipid management, a substantial proportion of high-risk patients do not achieve sustained LDL-C control in clinical practice. In such cases, early initiation of PCSK9 inhibitor therapy is clinically justified. These agents are effective not only in ASCVD but also in familial hypercholesterolaemia, where conventional therapies are often insufficient.

Current European Society of Cardiology guidelines recommend treatment intensification when LDL-C targets are not achieved despite maximally tolerated therapy. In this patient, LDL-C rose progressively to 248 mg/dL, exceeding the recommended target by more than fourfold, representing a marked increase in atherogenic burden and associated cardiovascular risk. The most striking feature of this case is the magnitude and rapidity of LDL-C increase despite apparently stable therapy. Such a change is unlikely to be explained by biological variability alone. Potential explanations include reduced medication adherence, analytical variability, or secondary dyslipidaemia. However, the patient reported adherence to therapy, and evaluation did not reveal thyroid dysfunction, renal impairment, electrolyte abnormalities, or poor glycaemic control, making secondary causes less likely. This supports the interpretation of true treatment failure and reinforces the need for prompt therapeutic escalation.

PCSK9 inhibitors, including Evolocumab and Alirocumab, provide a highly effective strategy for further LDL-C reduction. These monoclonal antibodies act by inhibiting PCSK9-mediated degradation of LDL receptors, thereby increasing hepatic clearance of LDL cholesterol (2,3). Alirocumab is a fully human IgG1 monoclonal antibody produced in Chinese hamster ovary cells, whereas Evolocumab is of the IgG2 isotype; however, both share the same mechanism of action targeting circulating PCSK9[2].

Both agents were approved in 2015 for use in adults with heterozygous familial hypercholesterolaemia or clinical ASCVD requiring additional LDL-C lowering beyond maximal-

ly tolerated statin therapy. Evolocumab additionally has an indication for homozygous familial hypercholesterolaemia[2]. Their efficacy and safety have been demonstrated in multiple randomised controlled trials. The FOURIER trial, involving 27,564 patients with ASCVD, showed that Evolocumab (140 mg every 2 weeks or 420 mg monthly) reduced LDL-C by approximately 59% and significantly decreased rates of non-fatal myocardial infarction and coronary revascularisation. Similarly, the ODYSSEY OUTCOMES trial, which included 18,924 patients following acute coronary syndrome, demonstrated that alirocumab (75–150 mg every 2 weeks) reduced major adverse cardiovascular events by approximately 21% when added to statin therapy, with the greatest benefit observed in patients who had not achieved LDL-C targets[1,3].

PCSK9 inhibitors are generally well tolerated, with injection-site reactions being the most common adverse effect, and no major safety concerns have been identified in clinical trials to date, although long-term surveillance remains necessary[2]. Their clinical benefit is most pronounced in patients at highest cardiovascular risk. Although a large proportion of ASCVD patients may theoretically be eligible for PCSK9 inhibitor therapy, widespread use is constrained by cost, making targeted use in very high-risk individuals the most cost-effective strategy[1].

Emerging therapies such as a small interfering RNA targeting PCSK9 synthesis, offer an alternative approach with less frequent dosing; however, long-term cardiovascular outcome data are still evolving, and monoclonal antibody PCSK9 inhibitors currently have the strongest evidence base.[4]

Overall, this case underscores the importance of close lipid monitoring and proactive treatment escalation. Delayed intensification in the presence of rising LDL-C may expose patients to preventable cardiovascular events. PCSK9-targeted therapies provide an effective means of achieving further LDL-C reduction and reducing cardiovascular risk in patients who remain above target despite standard therapy. Further research is needed to clarify long-term outcomes, including mortality, and to optimise their role within comprehensive secondary prevention strategies.

Conclusion

Marked LDL-C increase may occur in very high-risk ASCVD patients despite maximally tolerated statin–ezetimibe therapy, even in the absence of identifiable secondary causes. This pattern represents a clinically significant loss of lipid control and warrants prompt treatment escalation. Early recognition and initiation of PCSK9 inhibitor therapy are essential to restore lipid control and reduce the risk of recurrent cardiovascular events. Further research is needed to optimise management strategies in complex patients with multiple comorbidities.

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TABLES AND FIGURES

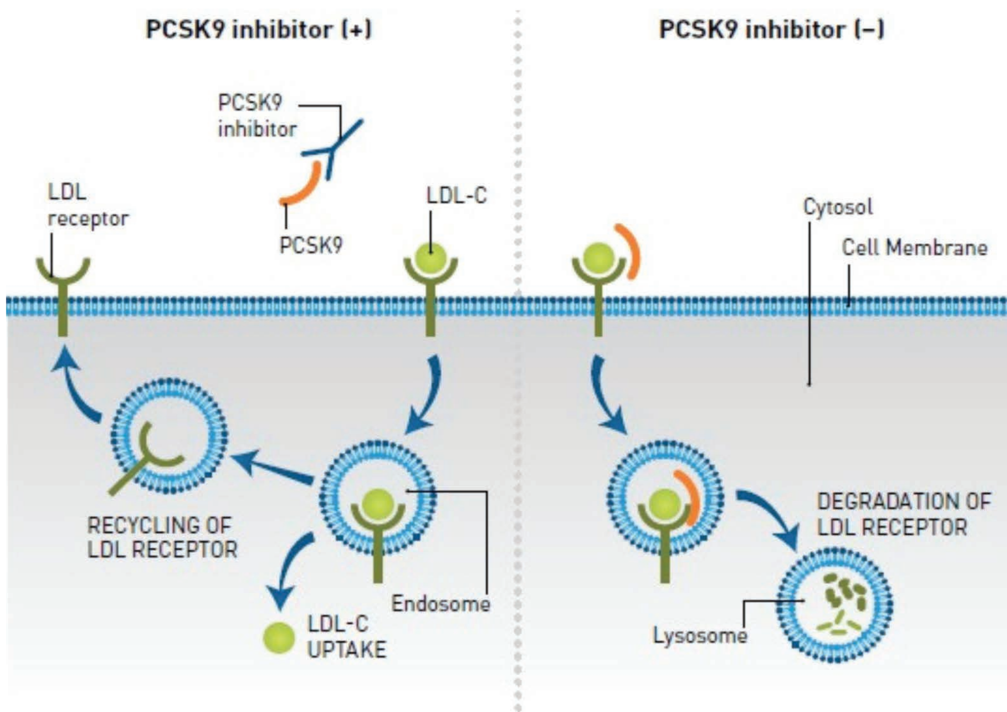

Figure 1. Mechanism of LDL-C reduction via PCSK9 inhibition. [5]

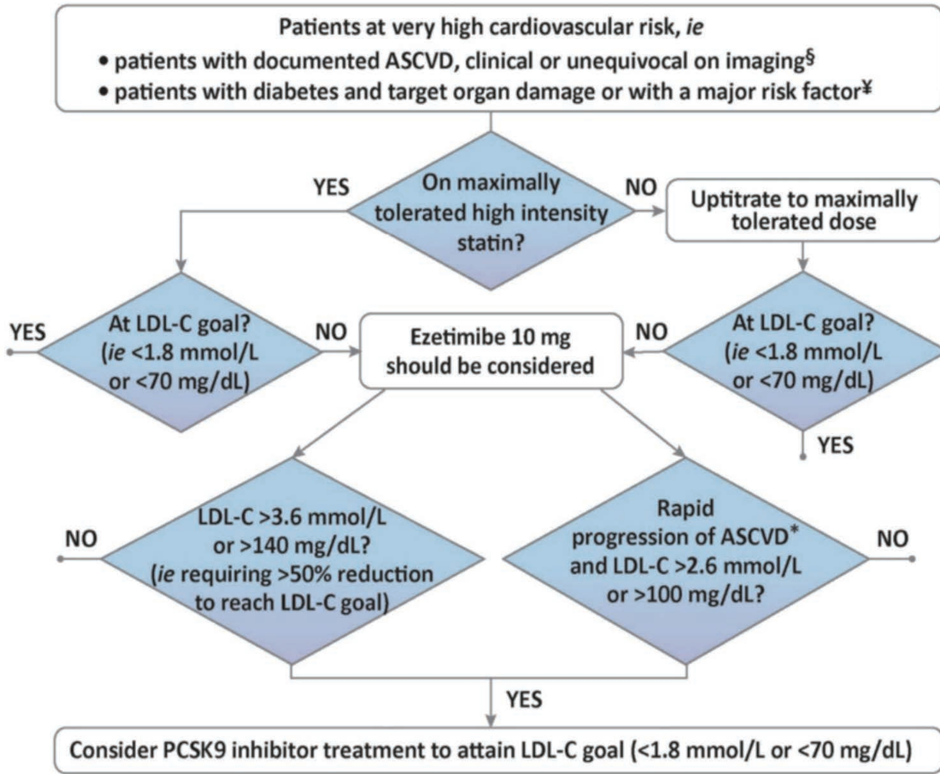

Figure 2. Treatment algorithm for lipid-lowering therapy in patients at very high cardiovascular risk.[1]

Difficulty evaluating the systolic heart function in a patient with heterotopic heart transplantation

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Introduction

Heterotopic heart transplantation is a surgical technique in which the donor heart is implanted alongside the native heart, allowing both organs to function in parallel1. In this configuration, the atria and great vessels of the donor heart are connected to those of the recipient, resulting in a unique dual-heart physiology[1]. Although this technique is rarely performed today, it remains an important surgical option in patients with pulmonary hypertension or donor–recipient size mismatch1. Evaluation of ventricular function in these patients may be challenging, as the coexistence of two hearts with different anatomical orientations and functional characteristics complicates echocardiographic imaging and interpretation3. We present a rare case of a patient with heterotopic heart transplantation referred for stress echocardiography, highlighting the technical and diagnostic challenges encountered during functional assessment[2,3].

Methods

A 45-year-old male with a history of heterotopic heart transplantation performed in 1997 for dilated cardiomyopathy, was referred to the echocardiography laboratory for stress echocardiography and coronary flow reserve assessment. His medical history included arterial hypertension, dyslipidaemia, hyperuricemia, coronary angiographies performed in 2000 and 2002 with non obstructive coronary artery disease. At presentation, the patient was hemodynamically stable and asymptomatic, with no significant abnormalities detected on physical examination. Baseline transthoracic echocardiography demonstrated the presence of both the native and transplanted hearts, creating technical challenges in image acquisition and interpretation. In parasternal long-axis and apical four-chamber views a cardiac structure with reduced contractility and significant valvular pathology was initially identified, corresponding to the native heart.

Further imaging in short-axis views revealed another cardiac structure with preserved sys-

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tolic function, consistent with the transplanted heart. Dobutamine stress echocardiography was performed using incremental infusion stages of 5, 10 and 20 mcg/kg/min in order to evaluate myocardial contractile reserve and detect inducible wall motion abnormalities.

Results

The examination proved technically demanding due to the complex anatomical configuration associated with heterotopic transplantation. Identification of the coronary arteries and reliable assessment of coronary flow reserve were not feasible during the study. Nevertheless, evaluation of the systolic response to inotropic stimulation was successfully performed. During dobutamine infusion, the transplanted heart demonstrated an appropriate increase in contractility and ejection fraction without new regional wall motion abnormalities. These findings indicated preserved myocardial contractile reserve and absence of inducible ischemia.

Conclusions

Heterotopic heart transplantation creates a unique dual-heart physiology that can significantly complicate echocardiographic evaluation of ventricular function, particularly during stress testing. Accurate interpretation requires careful anatomical identification of the native and transplanted hearts as well as an understanding of their distinct functional characteristics. Despite these challenges, stress echocardiography may still provide valuable information regarding myocardial contractile reserve when performed with meticulous imaging and interpretation. Awareness of the specific anatomical and physiological features of heterotopic transplantation is essential for accurate assessment and clinical decision-making in these rare patients.

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Bioprosthetic aortic and mitral valve degeneration treated with transcatheter valve in valve aortic and mitral valve implantation: a Valve-in-Valve TAVI and Valve-in-Valve TMVR case report

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Introduction

Transcatheter valve implantation has become a valuable treatment option for structural valve degeneration (SVD). Transcatheter Aortic Valve Implantation (TAVI) is established for aortic SVD, extending to younger and lower-risk patients. Transcatheter mitral valve replacement (TMVR) is emerging as an option for high-risk patients with failed mitral bioprosthesis, annuloplasty rings or severe annular calcification. With dedicated mitral devices in development, balloon-expandable prosthesis, used off-label for TMVR, demonstrate high technical success with favorable hemodynamic and clinical results is small series and registries data.

Concomitant aortic and mitral SVD presents a significant procedural challenge, esprescsially for high-risk patients. We report a fully transcatheter Valve-in-Valve (ViV) strategy combining TAVI and TMVR, as a less invasive alternative to repeat double-valve surgery, highlighting procedural aspects and short-term outcomes.

Methodology

This is a case report of a 76-year old female with severe symptomatic bioprosthetic aortic and mitral valve stenosis, following two double-valve replacements. Data were obtained from patient's medical file during peri-invasive assessment and hospitalization.

Results/ Case report

The patient, previously treated with double-valve (aortic and mitral) replacement (2015, 2021), presented with severe symptomatic stenosis of both bioprosthetises. Surgical risk was high (EuroSCORE II 19.9%) with complex comorbitities. ECG-gated-CT confirmed anatomical feasibility for transfemoral ViV-TAVI and ViV-TMVR. Thus, a 23mm self-expandable valve was implanted within

CITATION: Karmioti G., Eteocleous N., Zittis I., Myrianthopoulos K., Mitsis A., Eftychiou Ch., Ioannides M., Rotos Ch., Bioprosthetic aortic and mitral valve degeneration treated with transcatheter valve in valve aortic and mitral valve implantation: a Valve-in-Valve TAVI and Valve-in-Valve TMVR case report
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the previous aortic bioprosthesis, followed by a 26mm balloon-expandable valve implantation within the previous mitral bioprosthesis, yielding good hemodynamic results without procedural or vascular complications.

Conclusion

This case illustrates the feasibility of ViV procedures for both aortic and mitral SVDs. Detailed CT planning and valve selection is essential. While ViV-TAVI is well established, ViV-TMVR is emerging as feasible and safe when performed in selected patients.

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Cephalalgia as the Presenting Manifestation of Acute Coronary Syndrome

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Case description

An 84-year-old patient presented with a 3-hour history of new-onset headache that had awakened the patient from sleep. The headache was exertional and associated with nausea, without photophobia. Past medical history included coronary artery disease treated with PCI to the left main–left anterior descending artery and right coronary artery in 2020, as well as hypertension and dyslipidaemia.

Physical examination and brain CT were unremarkable. The ECG revealed ST-segment elevation in leads I and aVL with reciprocal ST-segment depression in II, III, and aVF *(Figure 1)*. Troponin was elevated at 38ng/mL (normal <0.10 ng/mL). Interestingly, resolution of the headache paralleled normalization of the ECG changes.

Echocardiography showed preserved left ventricular systolic function without regional wall motion abnormalities. Coronary angiography demonstrated a significant first diagonal branch stenosis, identified as the culprit lesion. PCI with a drug-coated balloon was successfully performed. The headache did not recur after revascularization. At 1 month, ECG showed only nonspecific lateral ST-T changes *(Figure 2)*.

Discussion

This case illustrates an unusual presentation of acute coronary syndrome (ACS), with headache as the predominant symptom. Cardiac cephalalgia is a rare but recognized manifestation of myocardial ischemia and may represent an ischaemic equivalent of angina. According to the International Classification of Headache Disorders (ICHD-3), it is typically characterized by new-onset headache, often triggered by exertion, occurring in the setting of myocardial ischemia, and improving after relief of ischemia.

In the present case, several findings supported this diagnosis: advanced age, cardiovascular risk factors, exertional headache, normal neurological examination and brain CT, ischemic

CITATION: Vassiliades A., Dimitriades V., StylianouV., Pavlou M., Georgiou P., Kapetis L., Patestos D., Tsielepis M., Bazoukis G., Cephalalgia as the Presenting Manifestation of Acute Coronary Syndrome CyJCardMed 2026, Volume 4, Issue 1, 39-41

ECG changes, marked troponin elevation, and complete resolution of symptoms after coronary revascularization. The temporal association between headache improvement and normalization of ECG abnormalities further strengthens the causal link.

The exact pathophysiology of cardiac cephalalgia remains uncertain. Proposed mechanisms include the release of vasoactive and inflammatory mediators during ischemia, as well as convergence between cardiac autonomic afferents and cranial pain pathways.

Although uncommon, headache may be the presenting symptom of ACS, particularly in elderly patients and in those with established cardiovascular disease. Awareness of this entity is clinically important, as failure to recognize cardiac cephalalgia may delay diagnosis and reperfusion, with potentially serious consequences.

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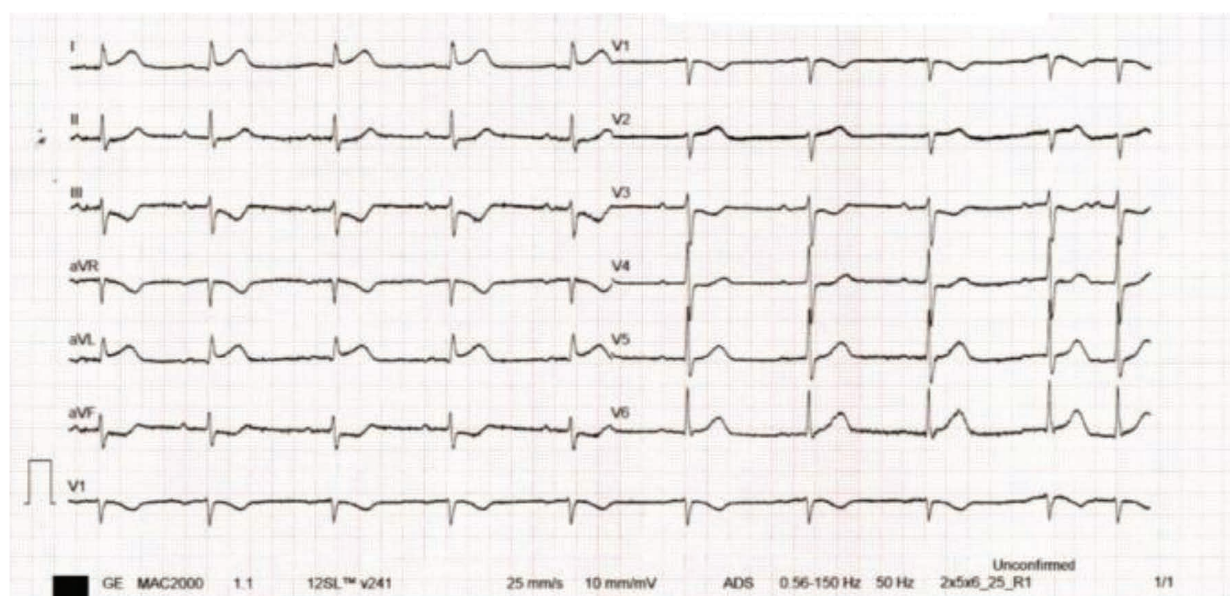

Figure 1. Presenting ECG showing ST segment elevation in leads I and aVL.

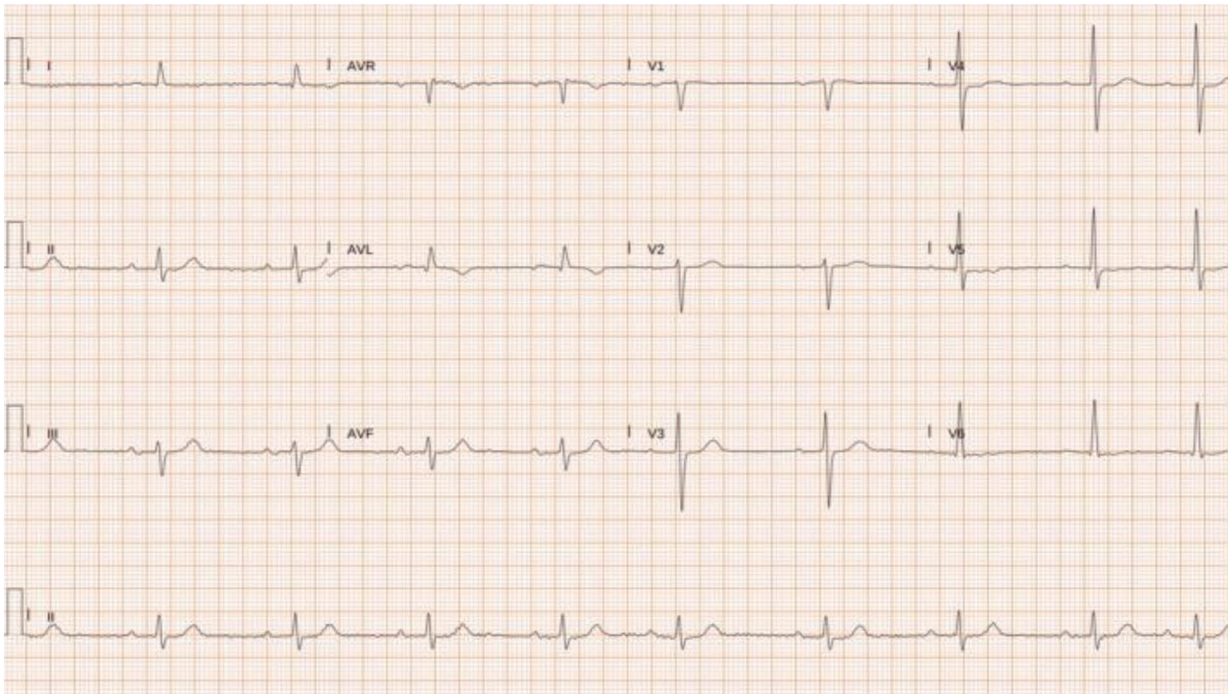

Figure 2. ECG one month post-PCI

Quantification of Aortic Stenosis in the Presence of Concomitant Subvalvular Obstruction and Mitral Regurgitation: Diagnostic Pitfalls and Multimodality Imaging Integration

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Introduction

Accurate quantification of aortic stenosis (AS) becomes particularly challenging in the presence of multilevel left ventricular outflow tract (LVOT) obstruction and concomitant mitral regurgitation (MR), as Doppler-derived gradients are highly flow-dependent and may be misleading. Historically, severity assessment relied predominantly on transvalvular velocities and gradients; however, in complex obstructive physiology this approach may result in misclassification and inappropriate therapeutic decisions. Integration of clinical findings with advanced multimodality imaging is therefore essential.

Methods

We report the case of a 52-year-old male presenting with exertional dyspnea and systolic murmur. Transthoracic echocardiography demonstrated elevated transaortic gradients with discordant parameters of AS severity. Transesophageal echocardiography identified a fixed subaortic membrane with additional dynamic LVOT contribution, while MR further altered forward flow conditions. Cardiac magnetic resonance imaging and invasive hemodynamic assessment were performed to clarify anatomical and functional severity at each obstruction level.

Results

Imaging confirmed combined valvular AS and subvalvular obstruction, resulting in multilevel LVOT stenosis. Doppler gradients alone overestimated the valvular component due to altered proximal velocities and flow redistribution. Three-dimensional transesophageal echocardiographic planimetry and multimodality integration enabled accurate anatomical quantification and differentiation of obstruction levels. The patient underwent extended

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septal myectomy, membrane resection, and aortic valve replacement with significant post-operative gradient reduction and symptomatic improvement.

Conclusions

In multilevel LVOT obstruction, reliance solely on Doppler-derived gradients and the continuity equation may lead to erroneous classification of AS severity. Comprehensive clinical evaluation combined with advanced multimodality imaging is crucial for accurate diagnosis and optimal management. Precise anatomical and hemodynamic characterization forms the basis of heart team decision-making and directly determines surgical strategy and patient outcomes.

Keywords: Aortic stenosis; Subaortic stenosis; Mitral regurgitation; Multimodality imaging; LVOT obstruction; Diagnostic Pitfalls.

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Complete Cardiac Recovery after Kidney Transplantation: A Case Report

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Introduction

Patients with end-stage kidney disease (ESKD) frequently develop left ventricular systolic dysfunction (LVSD) and functional mitral regurgitation (FMR). Although advanced heart failure may complicate transplant candidacy, emerging evidence suggests that renal transplantation can significantly improve cardiac structure and function.

Case report

A 47-year-old man with thin basement membrane nephropathy, diagnosed at age 33, developed progressive chronic kidney disease and reached end-stage renal disease by 2024, prompting evaluation for kidney transplantation. Pre-transplant cardiovascular assessment showed normal left ventricular size and systolic function (LVEF 60–65%) without valvular disease, and an exercise stress test was negative for inducible ischemia. These findings confirmed normal cardiac function prior to dialysis and transplantation. While awaiting transplantation, the patient was initiated on maintenance hemodialysis (December 2024), and a native arteriovenous fistula was created in January 2025, becoming functional by March 2025. Despite intensified dialysis, renal function continued to deteriorate. Clinically, he developed fatigue and palpitations, and in May a 24-hour Holter monitoring revealed a high burden of ventricular ectopy (~4,400 PVCs). Bisoprolol was initiated, resulting in partial symptomatic improvement.

Approximately two months later, the patient received an urgent call for kidney transplantation

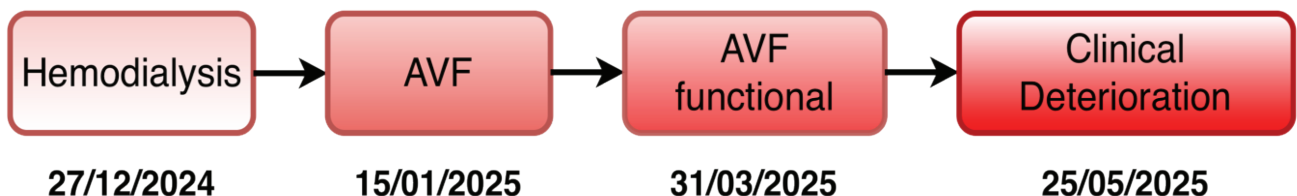

Figure 1. Clinical Timeline

CITATION: Kyriakou M., Ioannides M., Patsalou M., Yiangou K., Eftychiou Ch., Complete Cardiac Recovery after Kidney Transplantation: A Case Report CyJCardMed 2026, Volume 4, Issue 1, 44-47

from a deceased donor, prompting same-day cardiology reassessment. TTE revealed new-onset LV dilatation with diffuse hypokinesia and moderately reduced systolic function (LVEF ~40%), along with left atrial enlargement and moderate FMR representing the first evidence of LVSD. A clinical dilemma arose regarding whether to proceed with transplantation. Although ischemic evaluation was considered, the patient was young, asymptomatic, and had a previously negative stress test, while global hypokinesia favored a non-ischemic etiology. Given the time-sensitive nature of the graft and the likelihood of reversibility, transplantation was performed without additional cardiac testing. Kidney transplantation was performed the following day and was uneventful, with prompt improvement in renal function consistent with good graft function. However, shortly after discharge, the patient was readmitted with acute pulmonary edema, requiring urgent medical management.

At that time, a comprehensive cardiovascular evaluation was performed. Repeat TTE *(Figure 2)* demonstrated a severely dilated left ventricle (LVEDD 6.8 cm) with eccentric remodeling and moderately reduced systolic function (LVEF 35%), along with marked left atrial enlargement. Color Doppler showed an eccentric, posteriorly directed mitral regurgitation jet, consistent with secondary (functional) MR due to leaflet tethering (Carpentier IIIb). PISA-derived parameters suggested moderate MR (EROA 0.2 cm², regurgitant volume 37 mL), though severity was likely underestimated due to jet eccentricity and the Coanda effect.

Transesophageal echocardiography (TEE) *(Figure 3)* confirmed severe MR, with systolic pulmonary vein flow reversal.

The patient was decongested and discharged with a plan for stress cardiac magnetic reso-

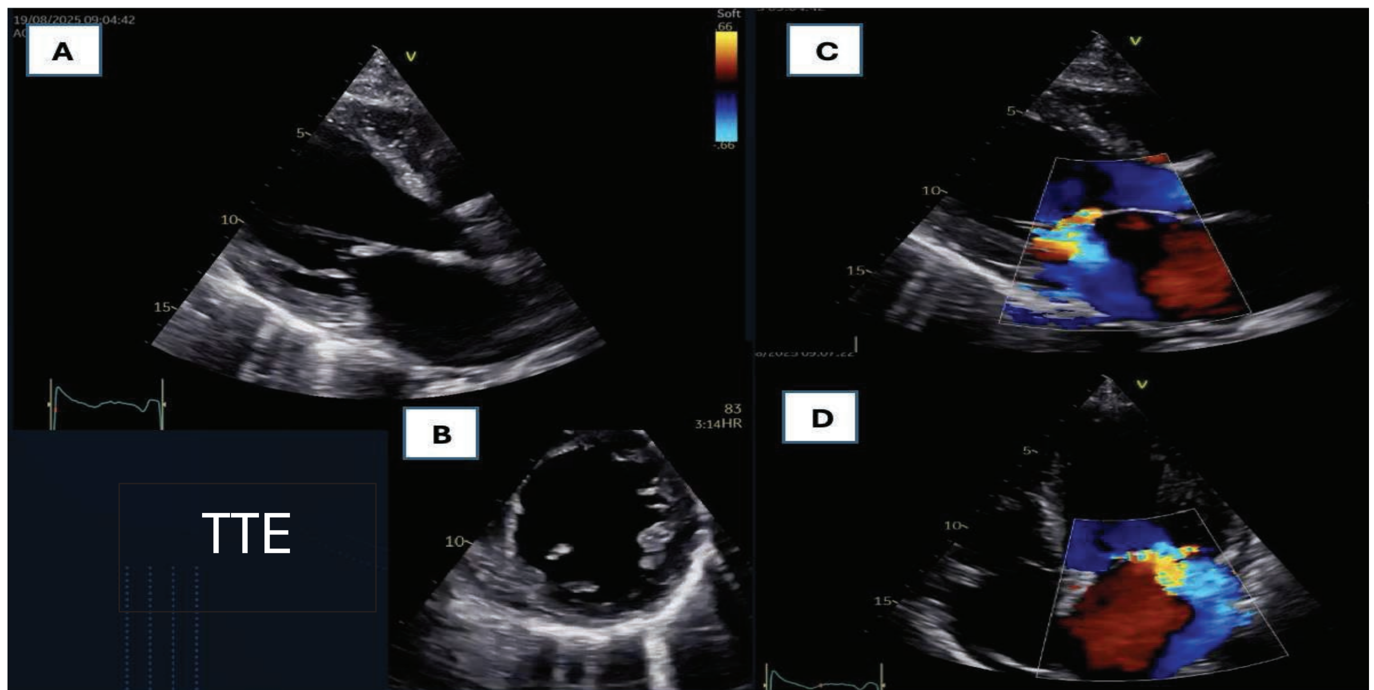

Figure 2. TTE demonstrates a severely dilated left ventricle in the parasternal long-axis (PLAX) (A) and short-axis (B) views. Color Doppler imaging reveals an eccentric, posteriorly directed mitral regurgitation jet in the PLAX (C) and apical four-chamber (D) views, consistent with FMR in the setting of LV dilatation and leaflet tethering.

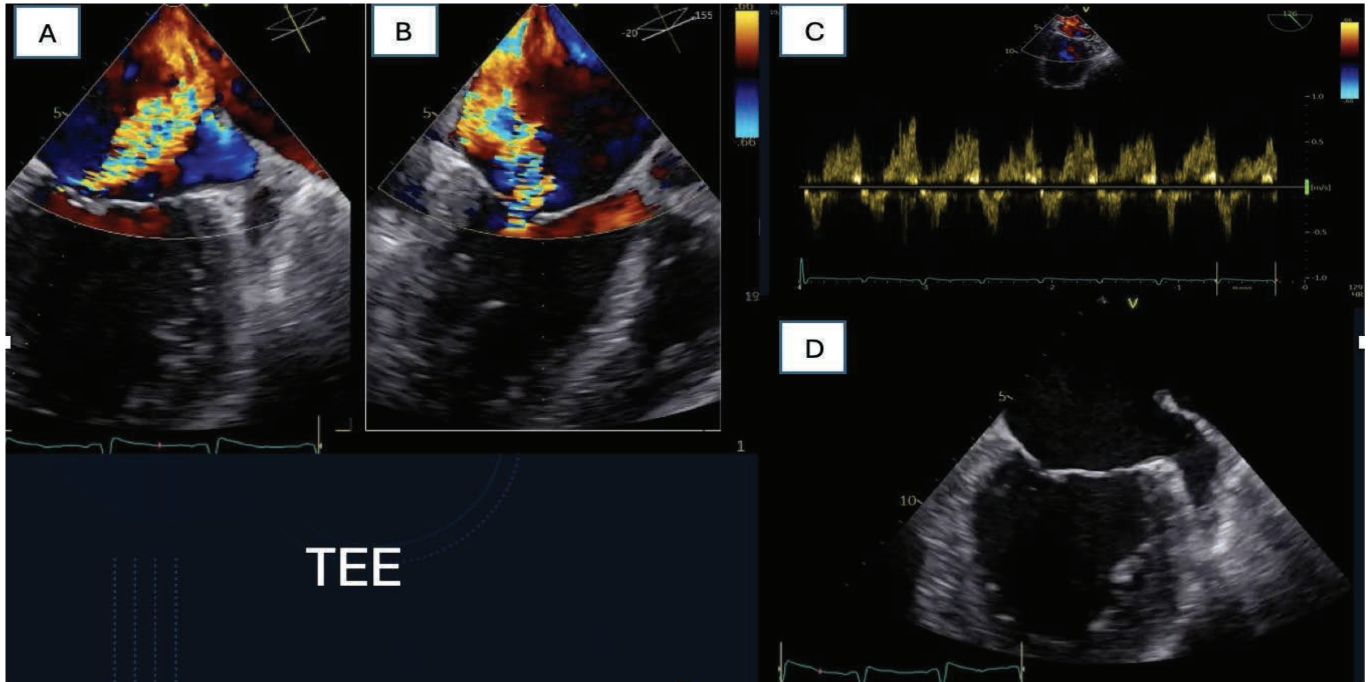

Figure 3. (A) Mid-esophageal (ME) commissural view with biplane cursor positioned at the A2 scallop. (B) Corresponding orthogonal long-axis view (A2–P2) with color Doppler demonstrating a large, eccentric mitral regurgitation jet directed posteriorly. (C) Pulsed-wave Doppler of the pulmonary veins demonstrating systolic flow reversal, confirming severe MR. (D) ME view illustrating LA and LV dilatation.

nance imaging (MRI) to assess for inducible ischemia and late gadolinium enhancement (LGE). Performed one month later, MRI showed a severely dilated left ventricle with mildly impaired systolic function and moderate functional mitral regurgitation, with no inducible ischemia and no LGE, supporting a diagnosis of non-ischemic dilated cardiomyopathy and suggesting a potentially reversible myocardial substrate.

Management was conservative, with optimization of heart failure therapy. Due to renal limitations, treatment included bisoprolol, empagliflozin, and furosemide, along with blood pressure control and fluid restriction. Renal function remained stable, and the patient improved clinically. Follow-up TTE, performed approximately five months after transplantation, demonstrated reverse remodeling, with reduced LV dimensions, improvement in LVEF to 60–65%, and regression of mitral regurgitation to mild.

Discussion

This case highlights the complex interplay between chronic kidney disease (CKD), heart failure (HF) and potentially reversible myocardial remodeling following kidney transplantation. Patients with advanced CKD frequently develop type 4 cardiorenal syndrome, in which chronic renal dysfunction contributes to progressive cardiac structural and functional abnormalities. Several mechanisms have been implicated, including chronic pressure and volume overload, activation of neurohormonal pathways (RAAS and sympathetic nervous system)[1], and the effects of uremic toxins, inflammation, oxidative stress, anemia, and disordered calcium–phosphate metabolism[2]. These factors collectively promote left ventric-

ular hypertrophy or dilatation, fibrosis, and diastolic and systolic dysfunction, ultimately leading to maladaptive remodeling[3].

In patients on hemodialysis, an arteriovenous fistula (AVF) represents an additional hemodynamic burden. AVF creation reduces systemic vascular resistance and increases venous return, leading to increased preload and cardiac output, which may ultimately result in left ventricular dilatation and heart failure[4].

Furthermore, our findings are supported by emerging evidence demonstrating the beneficial cardiac effects of kidney transplantation. A recent study by Salas-Pacheco et al. showed that kidney transplantation is associated with significant cardiac reverse remodeling, including a reduction in left ventricular mass, improvement in LVEF and global longitudinal strain (GLS), as well as improved diastolic function, reflected by lower E/e′ and reduced left atrial volume index[5]. These changes highlight the impact of restoring renal function on myocardial structure and performance, likely mediated by improved volume control, reduction in uremic toxins, and attenuation of neurohormonal activation.

Conclusion

Heart failure in advanced CKD may be reversible, especially in the absence of myocardial fibrosis. Kidney transplantation can promote reverse remodeling, emphasizing the importance of treating the underlying renal disease.

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Heart Failure in an elderly male with consideration of ICD upgrade to CRT-D

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Background
Heart failure with reduced ejection fraction (HFrEF) in elderly patients is frequently multifactorial and may be exacerbated by ventricular dyssynchrony in the case of a single or dual chamber device.

Case
We report the case of an 80-year-old male with ischemic cardiomyopathy, prior myocardial infarction, type 2 diabetes mellitus, and resected colorectal cancer who developed progressive HFrEF despite guideline-directed medical therapy (GDMT) and implantable cardioverter-defibrillator (ICD) implantation for primary prevention. Device interrogation demonstrated a high right ventricular (RV) pacing burden (97%) and an episode of non-sustained ventricular tachycardia. Echocardiography revealed severe left ventricular systolic dysfunction (EF<30%) with left bundle branch block morphology and marked QRS prolongation (180 ms). Following multidisciplinary discussion, a stress cardiac magnetic resonance imaging to assess lateral wall viability was recommended prior to considering upgrade to cardiac resynchronization therapy (CRT-D) pacemaker.

Discussion
This case highlights pacing-induced cardiomyopathy as a contributor to multifactorial HFrEF and underscores the importance of individualized, guideline-based decision-making in elderly patients with complex comorbidities.
Keywords: heart failure with reduced ejection fraction, cardiac resynchronization therapy, implantable cardioverter-defibrillator, pacing-induced cardiomyopathy, elderly, guideline-directed therapy

Introduction
Heart failure (HF) remains a major cause of morbidity and mortality worldwide, particularly in older adults in whom ischemic cardiomyopathy predominates [1]. Disease progression in this population is often multifactorial, driven by recurrent ischemia, metabolic comorbidities, and systemic stressors such as malignancy and major surgery.

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Guideline-directed medical therapy (GDMT) forms the cornerstone of HFrEF management, while device therapy including implantable cardioverter-defibrillators (ICDs) and cardiac resynchronization therapy (CRT) improves survival and functional status in selected patients[2]. However, chronic right ventricular (RV) pacing may worsen ventricular dyssynchrony and contribute to pacing-induced cardiomyopathy, for which CRT upgrade can be beneficial[3].

We present a case illustrating multifactorial HFrEF progression with suspected pacing-induced cardiomyopathy and consideration of CRT-D upgrade.

Case Presentation

An 80-year-old Cypriot male attended routine cardiology follow-up at Evangelismos Private Hospital (Paphos, Cyprus). His medical history included coronary artery disease with prior myocardial infarction treated by percutaneous coronary intervention (PCI), subsequent coronary artery bypass grafting (CABG), ischemic HFrEF (New York Heart Association class III), type 2 diabetes mellitus, and resected colorectal cancer.

Clinical Timeline

10 years prior: Myocardial infarction treated with PCI

8 years prior: Recurrent angina → CABG

5–6 years prior: Colorectal cancer resection; subsequent acute coronary syndrome treated with repeat PCI

2 years prior: Persistent HFrEF diagnosed; ICD implanted for primary prevention

Since ICD implantation, the patient experienced several hospitalizations for acute decompensated HF, predominantly with dyspnea, managed according to contemporary HF guidelines [1,4].

Current Assessment

The patient was clinically stable at follow-up.

Vital signs: heart rate 74 bpm, blood pressure 116/68 mmHg

Medications: sacubitril/valsartan, bisoprolol, spironolactone, empagliflozin, metformin, sitagliptin, ezetimibe/atorvastatin, rivaroxaban, pantoprazole, oral iron.

Laboratory testing demonstrated normocytic anemia with otherwise unremarkable biochemistry.

Electrocardiography showed sinus rhythm with RV-paced morphology and left bundle branch block pattern; QRS duration 180 ms.

Transthoracic echocardiography revealed severe LV systolic dysfunction (LVEF 30.7%), LV dilation (LVIDd 69 mm), and left atrial enlargement (52 mm).

ICD interrogation demonstrated 97% ventricular pacing and one episode of non-sustained ventricular tachycardia without therapy delivery.

Multidisciplinary Assessment

The combination of high RV pacing burden, prolonged QRS duration, LBBB morphology, and persistently reduced LVEF raised concern for pacing-induced cardiomyopathy.

Following presentation at a multidisciplinary team, the following strategy was agreed:

Optimize GDMT with addition of vericiguat to reduce recurrent HF decompensation risk.

Perform stress cardiac magnetic resonance imaging to assess inducible ischemia and myocardial viability in the lateral wall (target LV pac-

ing territory).

These assessments will guide the decision regarding potential upgrade to CRT-D.

Discussion

This case illustrates the complexity of managing multifactorial ischemic HFrEF in elderly patients with device therapy. Several mechanisms likely contributed to disease progression, including recurrent ischemia, metabolic comorbidity, and chronic RV pacing–induced desynchrony.

High RV pacing burden has been associated with worsening LV function and HF outcomes[3]. In patients with reduced LVEF, LBBB morphology, and substantial RV pacing, upgrade to CRT improves ventricular synchrony, functional capacity, and survival[5]. Current guidelines therefore support CRT in patients with LVEF ≤35% and high RV pacing burden or anticipated pacing dependence[2].

Pharmacologic optimization remains essential. Vericiguat has demonstrated reduced HF hospitalization risk in high-risk HFrEF populations and may improve stability prior to device intervention[4]. Additionally, myocardial viability assessment with cardiac MRI can help predict CRT response by confirming viable lateral wall myocardium suitable for LV lead placement.

The multidisciplinary approach in this case emphasizes individualized decision-making in advanced HF, particularly in elderly patients with multiple comorbidities.

Conclusions

Multifactorial progression of ischemic HFrEF in elderly patients may include pacing-induced cardiomyopathy from chronic RV pacing. Recognition of electrical dyssynchrony, optimization of GDMT, and viability-guided consideration of CRT-D upgrade are essential components of management. Structured multidisciplinary evaluation enables personalized, guideline-based therapy in complex heart failure cases.

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REVIEW ARTICLE

Arts Therapy Interventions in patients with Heart Failure: A Systematic Review

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Abstract

Aims: To evaluate the efficacy and safety of arts therapy interventions in patients with heart failure (HF), focusing on functional capacity, health-related quality of life (HRQoL), and psychological outcomes.

Methods and Results: A PRISMA-compliant systematic review was performed using MEDLINE, Embase, Cochrane CENTRAL, and PsycINFO (inception to December 2025). Randomized controlled trials and controlled interventional studies assessing music-based, dance/movement-based, or visual art therapies in adult HF patients were included. Primary endpoints were peak oxygen uptake (peak VO_2), six-minute walk distance (6MWD), and HF-specific HRQoL (Minnesota Living with Heart Failure Questionnaire [MLHFQ] or Kansas City Cardiomyopathy Questionnaire [KCCQ]). Random-effects meta-analysis was conducted when appropriate.

Eleven studies (n=612; mean age 66±7 years; 78% NYHA II–III) met inclusion criteria.

Dance-based interventions were associated with significant improvements in:

- Peak VO_2: +2.1 $mL \cdot kg^{-1} \cdot min^{-1}$ (95% CI 1.4–2.8; $p<0.001$)
- 6MWD: +38 m (95% CI 22–54; $p<0.001$)
- MLHFQ: −9.4 points (95% CI −13.1 to −5.7; $p<0.001$)

Music-based interventions improved HRQoL (SMD −0.46; 95% CI −0.70 to −0.22) and reduced depressive symptoms (SMD −0.39; $p=0.01$), without measurable effects on exercise capacity. Visual art therapy data were limited and not suitable for meta-analysis. No serious intervention-related adverse events were reported.

Conclusion: Arts therapy—particularly dance-based and music-based interventions—provides clinically meaningful improvements in functional capacity and HRQoL in stable HF patients and appears safe. These therapies may serve as effective adjuncts to guideline-directed HF care.

Introduction

Heart failure is characterized by persistent symptoms, reduced exercise tolerance, and impaired quality of life, even in patients receiving optimal guideline-directed medical therapy.

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Patient-reported outcomes, including HRQoL and functional capacity, are now recognized as independent predictors of hospitalization and mortality[1].

Beyond conventional cardiac rehabilitation, complementary non-pharmacological strategies that enhance adherence, motivation, and psychosocial well-being are increasingly relevant. Arts therapy—defined as structured therapeutic use of music, dance/movement, or visual arts—has shown benefit in other chronic conditions but has not been comprehensively evaluated within an HF-specific framework[2-5]. This systematic review aims to quantify the effects of arts therapy on clinically relevant HF outcomes.

Methods

Search Strategy and Study Selection

Databases searched included MEDLINE, Embase, Cochrane CENTRAL, and PsycINFO. Search terms combined heart failure with music therapy, dance therapy, movement therapy, and art therapy. Only studies enrolling adults with diagnosed HF were included.

Inclusion criteria

• Chronic HF (HFrEF)

• Arts-based intervention compared with usual care or standard rehabilitation

• Reported outcomes including peak VO_2, 6MWD, MLHFQ/KCCQ, or validated psychological scales

• Randomized or controlled interventional design

Statistical Analysis

Statistical analysis was performed using a random-effects model. Clinical relevance was interpreted using established HF thresholds (e.g. ≥5-point change in MLHFQ).

Results

Study Characteristics: Eleven studies were included with 6 randomized controlled trials and 5 prospective controlled studies. Most participants were NYHA class II–III with stable chronic HF. Intervention duration ranged from 8 to 24 weeks.

Dance-Based Therapy: Dance-based interventions (ballroom, traditional, or structured movement programs) consistently improved functional capacity and HRQoL.

• The pooled improvement in peak VO_2 (+2.1 $mL \cdot kg^{-1} \cdot min^{-1}$) exceeds the minimal clinically important difference reported in HF rehabilitation studies.

• Improvement in 6MWD (+38 m) suggests enhanced daily functional performance.

• MLHFQ reduction of −9.4 points represent a clinically meaningful and prognostically relevant improvement.

No increase in arrhythmias, hospitalizations, or exercise-related adverse events was reported.

Music-Based Interventions: Music-based interventions, primarily structured music listening, demonstrated moderate but consistent improvements in HRQoL and psychological outcomes.

• HRQoL improved with a pooled SMD −0.46, corresponding to a moderate effect size.

• Depression and anxiety scores were significantly reduced.

• No significant effect on peak VO_2 or 6MWD was observed.

These interventions were well tolerated and easily implemented in home-based HF care.

Visual Art Therapy: Only two small studies assessed visual art therapy in HF patients, mainly in advanced disease or palliative settings. Reported benefits included improved emotional expression, reduced distress, and better engage-

ment with care. Due to heterogeneity and limited sample size, quantitative synthesis was not feasible.

Discussion

This HF-focused systematic review demonstrates that arts therapy interventions produce measurable, clinically relevant benefits, particularly when physical movement is incorporated. Dance-based therapy appears comparable to conventional aerobic training in improving functional capacity while offering additional psychosocial benefits that may enhance long-term adherence[5].

Music-based interventions primarily affect HRQoL and psychological well-being—domains strongly associated with outcomes in HF but often insufficiently addressed in routine care[3].

Visual art therapy remains under-studied in HF and should currently be viewed as a supportive rather than rehabilitative intervention[5].

Clinical Implications: Arts therapy can be integrated into rehabilitation and chronic HF programmes and it is particularly suitable for older and frail HF patients, for patients with comorbid depression and anxiety and for patients with low adherence to conventional exercise. It aligns with ESC emphasis on patient-reported outcomes and holistic HF care[6].

Limitations: there are some limitations related to the short follow up duration, the lack of data on endpoints such as hospitalizations and mortality and the limited number of large - scale randomized trials.

Conclusions

Arts therapy interventions, especially dance-based and music-based programs, are safe and effective adjuncts in the management of stable heart failure. They improve functional capacity and quality of life beyond usual care and merit consideration within comprehensive HF management models. Further large-scale trials are required to define their long-term clinical impact.

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The European Exam in Core Cardiology

The European Exam in Core Cardiology (EECC) previously known as European Exam in General Cardiology (EEGC), is administered by the European Society of Cardiology, in conjunction with the UEMS Cardiology Section and the National Cardiac Societies.

Passing the EECC is a curriculum requirement in some countries and for the CCT in the UK.

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Once registration is closed, the European Society of Cardiology (ESC) will provide access to preparatory modules for the exam via the ESC e-learning platform to everyone registered for the EECC. To do this, candidates need to supply their contact details to the ESC.

The Exam takes place once a year in June. This year it was organized on the 18th of June. The date for the next year will be Tuesday 17 June 2025.

The registration is managed by the Cyprus Society of Cardiology and usually starts in September and closes in February the next year. The exam is open only to cardiology trainees who are on their third or later year of specialty training.

The following information is necessary for registration:

- Email address
- ID number
- Phone number

Candidates receive a confirmation e-mail from the ESC.

Exam preparation

The EECC is designed to test a wide range of cardiology knowledge aligned with the ESC core curriculum. There is no single source from which the questions are developed, and candidates are encouraged to read the ESC guidelines, the ESC textbook and practice-changing clinical trials.

Registering for EECC gives access to a preparatory course, designed to support candidates in their preparation for the exam. A mock exam of 60 MCQs will be made available to candidates one month before the exam.

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The following modules are part of the EECC.

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Module 3. Valvular heart disease
Module 4. Rhythm disorders
Module 5. Heart Failure
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