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EDITORIAL

The challenge of management of arterial hypertension

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Introduction

Arterial hypertension (AHT) represents an important risk factor for cardiovascular disease and stroke [1]. Its prevalence in the general population is approximately 40%, with a higher prevalence in elderly and lower in younger individuals [2]. Approximately half of the hypertensive patients are sub-optimally treated, [3], leading to increased risk for complications and mortality. In general, a blood pressure above 120/80 may be considered as the cutting point for introduction of therapeutic measures, targeting to lower levels. Lifestyle behaviour and pharmacotherapies are both important components of the overall management of the hypertensive patient. These are briefly presented in this editorial.

Healthy Lifestyle

The reduction of sodium intake, to less than 2.5 g/day, the addition of potassium intake to more than 3.5 g/day, and the use of Mediterranean and DASH diet, are considered effective components for hypertension management. In addition, regular exercise and weight loss in the overweight and obese persons, may reduce the risk for hypertension and can help normalizing blood pressure in hypertensive persons [4]. Other healthy lifestyle measures, include smoking cessation [9], reduction of alcohol consumption [10] and avoidance of sugary and energy drinks [11], which are related not only with increased levels of blood pressure but also with other morbidities such as diabetes and cancer.

Pharmacotherapy

The 2024 ESC guidelines on AHT recommend a combined approach of healthy lifestyle behaviour and medical therapy for managing hypertension [4]. On the first line of treatment for AHT are the ACE-inhibitors and the angiotensin II receptor blockers (ARBs), the dihydropyridine calcium channel blockers (CCB), and the diuretics, preferably of the thiazide family. Patients usually need two or more agents from different groups, for appropriate management. Patient adherence to therapy is a reality, but it seems that a single-pill combination, may overcome the problem and enhance patient adherence [3,4]. Additional medicines given in resistant

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AHT are alpha blockers, centrally acting agents and aldosterone antagonists. Beta blockers such as atenolol, metoprolol and bisoprolol are useful in patients with history of coronary artery disease, chronic heart failure and arrhythmias, whereas nebivolol has a significant antihypertensive effect rather than antianginal, antiarrhythmic or anti-failure, explained partly by an increased release of nitric oxide [12,13].

Ambulatory and Home BP Measurement

A major problem in clinical setting is the phenomenon of white-coat hypertension, which prevents accurate measurements of BP. This is largely overcome by the home blood pressure measurements (HBPM) and the 24-hour ambulatory blood pressure measurements (24hABPM). The latter may give, in addition, information on the oscillations of BP at night, allowing clinicians to understand better the circadian and the nocturnal dipping patterns of BP [5]. Moreover, the BP variability, assessed by the 24hABPM, allows modification of BP management, since it is known that variability is an independent risk for cardiovascular disease [6].

Resistant Hypertension

Some patients continue to have elevated BP despite healthy lifestyles and medical treatment from at least three different pharmacological groups in optimally tolerated doses. These patients are considered to have resistant hypertension and may account for a percentage ranging from 6 to 18% [7]. Guidelines suggest that these patients may benefit from the use of aldosterone antagonists (spironolactone, eplerenone) or if these are contraindicated or not well tolerated, then alpha blockers, vasodilators or centrally acting agents, such as doxazosin, clonidine, methyldopa or hydralazine, can be prescribed. Renal denervation may be considered as a final step, in difficult cases, with resistant hypertension, on maximal tolerated medical therapy [8].

Conclusion

The management of arterial hypertension, the most frequently clinically met disease and an important cardiovascular risk, has many challenges. Healthy lifestyle patterns along with optimal medical pharmacotherapy, are necessary for an effective management. Home and 24h ambulatory BP measurements may be needed to overcome the problem of white-coat hypertension and to assess the circadian and the night oscillations of blood pressure. Renal denervation may be useful in resistant hypertension, despite maximally tolerated therapy. There is however a long way to cover in understanding and managing AHT and in reducing its consequences.

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ABSTRACT 1 CARDIOLOGY CONFERENCE - 2025

Transvenous Cardiac Implantable Electronic Device Removal: the experience of the Nicosia General Hospital

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Introduction

Cardiac Implantable Electronic Device (CIED)-related infections are associated with high morbidity and mortality rates, necessitating timely and effective removal of the affected system. The dataset presented refers to the 7-year experience of the Nicosia General Hospital, and its dedicated multidisciplinary team.

Methods

Registry analysis by 4 reviewers, incorporating data from multiple patient records.

Results

From August 2018 to February 2025, 43 procedures were performed (10 explants, 33 extractions) for CIED-related infections, in 40 patients (36 males, 4 females, mean age 74 years), with increasing rate following the introduction of the Cyprus Healthcare System. Pocket dehiscence was the commonest indication (lead endocarditis rate approximately 26%). Common patient comorbidities include diabetes and hypertension. A total of 89 leads were removed (60% pace/sense, 27% defibrillation - 58% dual-coiled, and 13% left ventricular leads), from different manufacturers (69% Medtronic, 18% St Jude/Abbott), with 70% of active fixation mechanism (median lead age of 6 months for explants and 6 years for extractions). The average hospital inpatient stay was 34 days. Staphylococcus species (20% Epidermitis, 12% Methicillin-Sensitive Aureus) were the commonest microbial isolates. Serious, peri-procedural complications included 1 death due to SVC tear (2% procedural risk) and one cardiogenic shock due to acute right ventricular failure. Long-term follow-up revealed one-month mortality at 10%, and 5 patients (14%) did not proceed with re-implantation.

Conclusions

This is the first formal reporting for this clinical discipline in Cyprus, and highlights the low

CITATION: Sokratous St., et al, Transvenous Cardiac Implantable Electronic Device Removal: the experience of the Nicosia General Hospital, CyJCardMed 2025, 3, 1, 5-6.

periprocedural risk in our experience, whilst emphasizing the need for expanding this registry on a national reporting basis.

Keywords: pacemaker, cardiac implantable devices, extraction, endocarditis

ABSTRACT 2 CARDIOLOGY CONFERENCE - 2025

Cardiac Papillary Fibroelastoma Presenting with Cerebral and Myocardial Emboli

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Background

Cardiac papillary fibroelastoma (CPF) is a rare benign tumor that can serve as a source of embolism, leading to cerebrovascular and myocardial events.

Case Presentation

A 32-year-old male smoker with a family history of coronary artery disease presented with a one-week history of impaired focus in the left eye and upper quadrantic hemianopsia. Brain MRI revealed ischemic changes in the right orbital region, while angiography demonstrated stenosis of the right spinal and median cerebral arteries. Initial investigations, including a bubble study, 24-hour Holter monitoring, and carotid/spinal artery Doppler, were unremarkable aside from hypercholesterolemia managed with high-intensity statin therapy. Thrombophilia screening was also negative. Transthoracic echocardiography (TTE) showed regional wall motion abnormalities in the inferior and inferolateral segments of the left ventricle, corresponding with electrocardiographic findings of ischemia. Subsequent transesophageal echocardiography identified a hyperechogenic mass measuring 0.8 x 0.6 cm on the atrial surface of the A3 scallop of the mitral valve, suggestive of CPF. Coronary angiography revealed mildly atheromatous coronaries without significant stenosis. Cardiac magnetic resonance imaging (cMRI) confirmed the presence of a fibroelastoma and additionally detected multiple foci of myocardial scarring of ischemic etiology, without evidence of acute injury.

Management

The findings support a diagnosis of embolic ischemic stroke and silent myocardial infarctions secondary to CPF. The patient is scheduled for surgical resection of the lesion.

Conclusion

This case underscores the importance of a comprehensive cardiac evaluation in young patients presenting with embolic phenomena, as early detection of CPF can lead to timely surgical intervention and better clinical outcomes.

Keywords: ischemic stroke, myocardial infarct, embolism , cardiac papillary fibroelastoma

CITATION: Kyriakou M., et al, Cardiac Papillary Fibroelastoma Presenting with Cerebral and Myocardial Emboli, CyJCardMed 2025, 3, 1, 7.

ABSTRACT 3 CARDIOLOGY CONFERENCE - 2025

The Hidden Culprit: Coronary Embolism as a Rare Cause of Acute Myocardial Infraction

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Introduction

Acute myocardial infarction (AMI) is a life-threatening condition typically caused by atherosclerotic plaque rupture, followed by intracoronary thrombus formation. However, in rare cases, AMI may be attributed to embolic events, resulting in coronary embolism (CE). CE is recognized as a cause of myocardial infarction with non-obstructive coronary arteries (MINOCA) and should be suspected in cases of high thrombus burden despite a relatively normal underlying vessel. We present a unique case of AMI caused by an embolic event from a prosthetic mechanical aortic valve in the setting of suboptimal warfarin dosage and a subtherapeutic INR. This case underscores the importance of early recognition, multimodal imaging, and tailored anticoagulation strategies to optimize outcomes in high-risk patients.

Methods

We describe the case of a 58-year-old male with a known history of aortic valve replacement via the Bentall procedure, who presented to the Emergency Department (ED) with anterior ST-elevation myocardial infarction (STEMI). Urgent coronary angiography revealed a mid-left anterior descending artery (LAD) bridge, occluded by a large thrombus, with additional thrombus material present in the intermediate branch (IB). Intravascular ultrasound (IVUS) confirmed the presence of an intracoronary thrombus without significant underlying atherosclerosis in mid-LAD bridge. Initial management included thromboaspiration and angioplasty with a drug-eluting balloon (DEB). The patient was subsequently started on intravenous glycoprotein IIb/IIIa inhibitors, dual antiplatelet therapy, followed by a staged coronary angiogram during hospitalization to assess thrombus resolution.

Results

The patient remained hemodynamically stable, with progressive symptom relief. Serial electrocardiograms showed persistent ST-elevation in

CITATION: Ioannou M., et al, The Hidden Culprit: Coronary Embolism as a Rare Cause of Acute Myocardial Infraction, CyJCardMed 2025, 3, 1, 8-9.

the precordial leads. Final echocardiography before discharge revealed a left ventricular ejection fraction (LVEF) of 40%, with akinesis of the apex and anterior segments, but no evidence of left ventricular thrombus. A staged coronary angiography with IVUS reassessment confirmed thrombus resolution and patency of the LAD and IB arteries. Medical therapy was optimized, including anticoagulation with an appropriate warfarin dosage, antiplatelet therapy, and lipid-lowering agents, with instructions for frequent INR monitoring.

Conclusions

This case highlights the need for a high index of suspicion for CE in patients with prosthetic mechanical valves, even in the presence of therapeutic anticoagulation. Multimodal imaging, including IVUS, played a crucial role in diagnosis and management. A tailored therapeutic approach is essential for optimizing outcomes in these cases.

Keywords: coronary embolism, acute myocardial infarction, prosthetic mechanical valves, warfarin, intravascular ultrasound.

ABSTRACT 4 CARDIOLOGY CONFERENCE - 2025

Reverse Takotsubo Syndrome: A Surprising Sequel to Postoperative Anaphylaxis

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Introduction

Takotsubo syndrome (TTS), also known as stress-induced cardiomyopathy, is a transient cardiac condition that mimics acute coronary syndrome but occurs in the absence of significant coronary artery disease. It is typically triggered by extreme emotional or physical stress and is characterized by transient left ventricular dysfunction with wall motion abnormalities. While classic TTS predominantly affects the apical segments of the left ventricle, the less common reverse Takotsubo syndrome (rTTS) primarily involves the basal segments, with apical hyperkinesis. Its pathophysiology is not fully understood, but it is believed to involve catecholamine-induced myocardial stunning, abnormal sympathetic activation, and microvascular dysfunction. Anaphylaxis is a life-threatening condition that can lead to profound haemodynamic changes and intense catecholamine surges, which predispose patients to stress-induced cardiomyopathy. We present a case of reverse Takotsubo syndrome following severe postoperative anaphylaxis, highlighting the importance of early recognition and appropriate management.

Methods

We report a case of a 40-year-old female who, during gynaecological surgery, developed anaphylaxis triggered by antibiotic and tranexamic acid administration, prompting an urgent cardiology consultation due to perioperative arrhythmias. During the evaluation, echocardiography revealed severe left ventricular dysfunction (LVEF 20-25%), with basal and mid-segment hypokinesis and preserved apical function, findings characteristic of Reverse Takotsubo Syndrome (rTTS). Following surgery, the patient was admitted to the Cardiology Care Unit for further evaluation and management. Coronary angiography confirmed unobstructed coronary arteries, and 24-hour Holter monitoring demonstrated frequent ventricular ectopic beats but no sustained arrhythmias. The patient was administered heart failure-directed medical therapy, leading to gradual improvement of cardiac function.

CITATION: Leonidou E., et al, Reverse Takotsubo Syndrome: A Surprising Sequel to Postoperative Anaphylaxis, CyJCardMed 2025, 3, 1, 10-11.

Results

The patient remained hemodynamically stable, and serial echocardiographic follow-up demonstrated progressive left ventricular recovery, with LVEF returned to 50-55% and global longitudinal strain (GLS) of -14%. She was discharged with instructions to remain on prescribed anti-HF medication. Ten days after the event, Cardiac Magnetic Resonance Imaging (CMR) confirmed normal ventricular volumes, absence of myocardial fibrosis, no infarction or edema, and LVEF recovery to 57%, reinforcing the transient and reversible nature of stress-induced cardiomyopathy.

Conclusions

Reverse Takotsubo Syndrome (rTTS) should be recognized as a potential complication of severe allergic reactions and perioperative stress. This case highlights the critical importance of early recognition, multimodal imaging and close clinical follow-up to ensure accurate diagnosis, appropriate management and differentiation from other forms of acute cardiomyopathy.

Keywords: Reverse Takotsubo, stress-induced cardiomyopathy, ventricular tachycardia, postoperative anaphylaxis, transient myocardial dysfunction.

ABSTRACT 5 CARDIOLOGY CONFERENCE - 2025

Audit on Achieving Target Blood Pressure Based on Pharmacological Treatment in Hypertensive Patients According to 2024 European Society of Hypertension Guidelines

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Background

Hypertension remains a leading cause of cardiovascular morbidity and mortality worldwide, with only 21% of affected individuals achieving adequate blood pressure (BP) control. The 2024 European Society of Hypertension (ESH) guidelines emphasize individualized BP targets and the early use of combination therapy to optimize hypertension management. This audit evaluates BP control among hypertensive patients at Evangelismos Private Hospital to assess adherence to these guidelines. The audit was supervised by dr Joseph Moutiris.

Methods

A mixed-method audit was conducted, enrolling 20 consecutive hypertensive patients visiting the Cardiology clinic of Evangelismos Hospital, Paphos, in September 2024. Patients were assessed for BP target achievement (<140/90 mmHg for most; <130/80 mmHg for high-risk individuals) within three months of treatment initiation. The use of pharmacological therapy, including monotherapy versus combination therapy, was analyzed in relation to treatment success.

Results

- **BP Control:** 50% of patients achieved target BP levels; 45% failed to meet targets within the recommended timeframe.
- **Combination Therapy:** Initiated in 30% of patients; however, only 33% of these achieved BP control, suggesting suboptimal drug selection or patient adherence issues.
- **Delayed Achievement:** 50% of patients required more than three months to reach target BP, emphasizing the need for enhanced follow-up and therapy adjustments.

Conclusion

While half of the patients achieved BP targets, the delayed response and suboptimal use of combination therapy indicate a gap in adherence to ESH guidelines. Strengthening follow-up protocols, optimizing treatment regi-

CITATION: Sachdev Pr., et al, Audit on Achieving Target Blood Pressure Based on Pharmacological Treatment in Hypertensive Patients According to 2024 European Society of Hypertension Guidelines, CyJCardMed 2025, 3, 1, 12-13.

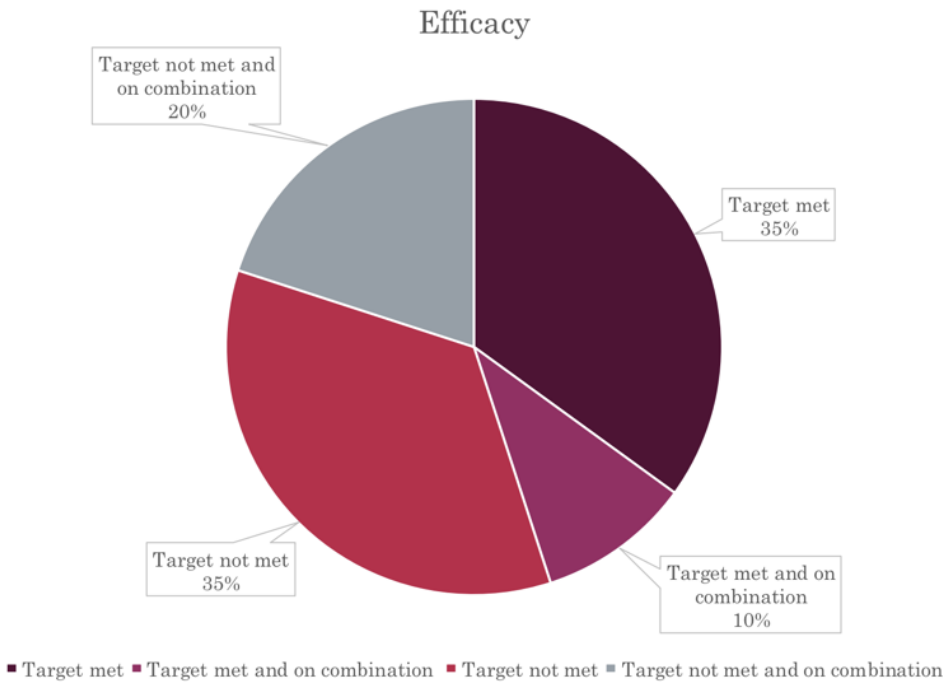

Figure 1. *Efficacy of Antihypertensive Treatment: 35% of Patients Achieved Target Blood Pressure, while 10% Achieved Target on Combination Therapy. However, 35% Did Not Meet the Target, and 20% Remained Uncontrolled Despite Combination Therapy."*

mens, and addressing medication adherence could enhance patient outcomes. A second audit cycle is necessary to measure the impact of these interventions.

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

ORIGINAL CLINICAL ARTICLE

Dance Movement Therapy and Depression in patients with chronic Heart Failure

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Introduction

Heart failure (HF) is a chronic and progressive condition characterized by the heart's inability to pump sufficient blood to meet the body's needs. It affects over 55 million people globally, a dramatic rise from 25.4 million in 1990, nearly doubling over three decades [(1)]. In addition to its physiological burdens, heart failure is closely linked with psychological comorbidities, particularly depression, which affects approximately 41% of patients with HF [(2)]. Depression in HF is associated with poorer prognosis, increased hospitalizations, and reduced quality of life. Among various non-pharmacological interventions, movement therapy (physical activity interventions) has emerged as a promising approach in the management of HF in reducing depression and improving the quality of life.

Definition of Dance Movement Therapy (DMP) and Methods

DMP is defined as "the psychotherapeutic use of movement to promote emotional, social, cognitive, and physical integration of the individual" [(3)].

This is a mixed method study, composed of both quantitative and qualitative data. For the collection of the quantitative data, the Minnesota Living with Heart Failure questionnaire and the Greek version of the Hospital and Anxiety Depression scale (HADS), were used. The qualitative data were exclusively based on patents' descriptions.

Patient characteristics

Dance Movement Therapy was applied for 12 months to twelve patients, in Paphos General Hospital, in 2021-2. All patients had chronic heart failure and reduced ejection fraction less than 40%. All patients were on optimal anti-failure treatment based on the most recent ESC Guidelines and six patients had also pacemaker, an ICD or a CRTd. Ten patients were men and two women. Mean age was 70±12 years. History of coronary artery disease had six patients, history of dilated cardiomyopathy had 2 patients and the rest had history of valve disease.

The degree of depression, was assessed before initiation of the dance movement therapy sessions, at six and at 12 months.

CITATION: Mchitarian M., et al, Dance Movement Therapy and Depression in patients with chronic Heart Failure, CyJCardMed 2025, 3, 1, 14-17.

Patient	*Baseline*	*At 6 months*	*At 12 months*
1	17	10	5
2	16	10	1
3	21	9	2
4	2	1	0
5	15	9	2
6	7	4	2
7	13	11	6
8	5	4	2
9	18	11	10
10	3	1	1
11	19	5	5
12	6	5	5

0 – no depression,
21-maximal degree of depression

Table 1. *Degree of depression at baseline, at 6 and 12 months after dance movement therapy*

Results

The results are shown in the table. Briefly, there was a decrease in the degree of depression in all 12 patients studied, at 6 months and this beneficial effect was maintained through the 12th month.

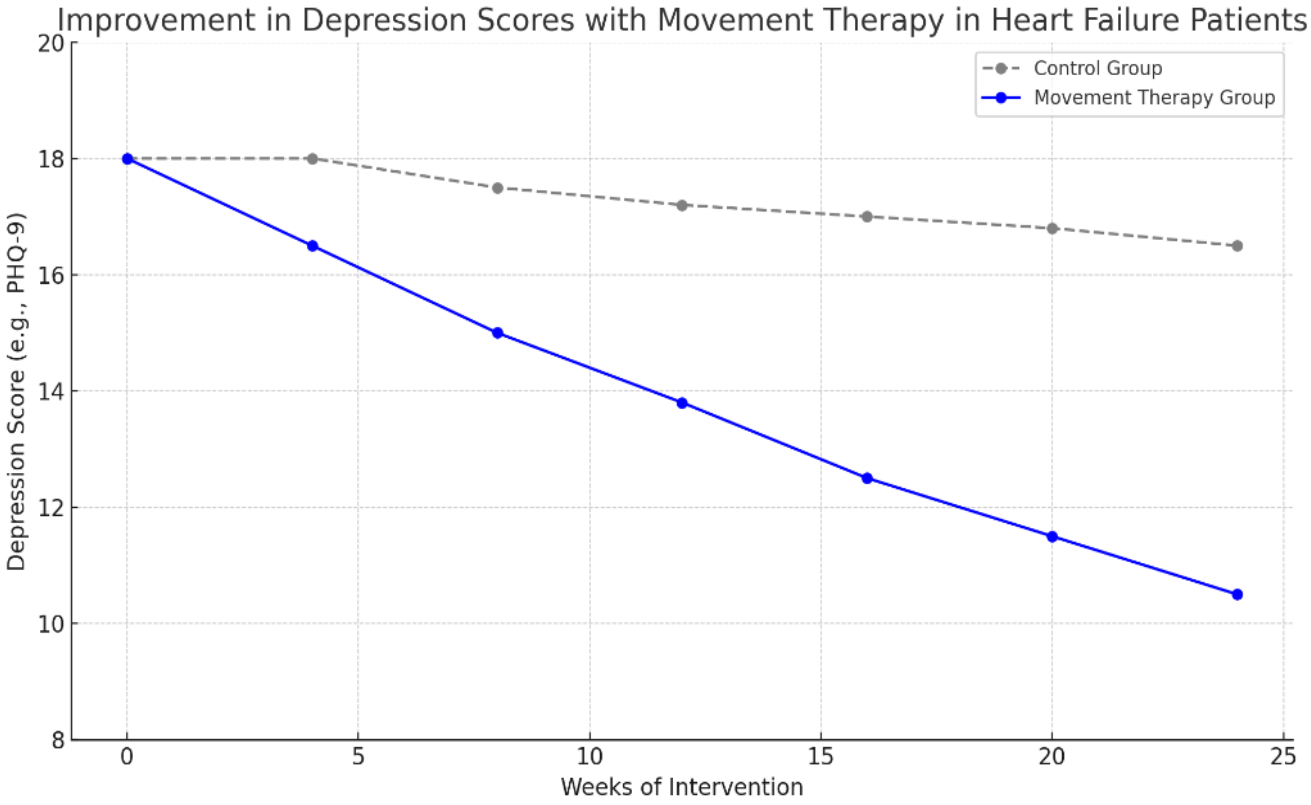
Discussion

The relationship between heart failure and depression is well documented. Depression may arise due to physical limitations, lifestyle restrictions, and the neurohormonal and inflammatory changes associated with HF [4] but also because of the fear for death. Conversely, depression can exacerbate HF outcomes through poor adherence to medication and self-care, unhealthy behaviour, and an increased sympathetic activity and inflammation [5]. Thus, addressing depression is essential in the overall care of HF patients.

Dance Movement therapy includes aerobic exercise, resistance training, yoga, and other structured physical activities. It has demonstrated positive effects on both cardiovascular and mental health outcomes in HF patients.

Exercise improves cardiovascular function by enhancing endothelial function, reducing inflammation, and improving skeletal muscle metabolism [6]. Psychologically, it acts as a natural antidepressant by stimulating the release of endorphins and neurotransmitters such as serotonin and dopamine. Additionally, exercise provides a sense of control, achievement, and social interaction—all crucial for improving mood in chronically ill individuals.

Several randomized controlled trials have shown that movement therapy significantly reduces depression symptoms in HF patients. A meta-analysis by Huang et al [7] found that exercise interventions reduced depressive symptoms by 31% in HF patients. The HF-ACTION trial, a large-scale study, demonstrated that regular aerobic exercise led to modest improvements in depression and functional capacity [8]. Moreover, mind-body exercises such as yoga and tai chi have also shown beneficial effects. Yeh et al. [9] found that tai chi improved mood and quality of life in patients with chronic systolic heart failure, in addition to enhancing exercise capacity.



Source: *Huang, K., Liu, W., He, D., Huang, B., & Xiao, D. (2019). Exercise training effects on depression in patients with chronic heart failure: A meta-analysis of randomized controlled trials. European Journal of Cardiovascular Nursing, 18(4), 260–269. https://doi.org/10.1177/1474515119835717*

Despite its benefits, implementing movement therapy in HF patients is not without challenges. Fatigue, fear of symptom exacerbation, lack of motivation, and physical comorbidities may limit participation. Therefore, programs must be tailored to individual capabilities, and healthcare providers should emphasize gradual, supervised activity progression.

Psychological readiness and support are also crucial. Combining movement therapy with counselling or cognitive behavioural therapy may enhance adherence and effectiveness, especially in severely depressed patients [10].

Our study showed that Dance Movement Therapy is able to reduce the degree of depression and maintains this effect up to 12 months.

Conclusion

Dance Movement Therapy represents a safe and effective strategy addressing depression in patients with heart failure. It is associated with improved mental health outcomes, when added to optimal medical therapy. As the burden of HF and depression grows, incorporating dance movement therapy into standard care, may offer additional and sustainable improvements in the quality of life of patients.

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CLINICAL ARTICLE

The value of genetic testing in Hypertrophic Cardiomyopathy

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Introduction

Cardiomyopathies are the most common cause of sudden death in young individuals. Hypertrophic cardiomyopathy (HCM) is the most frequent form of cardiomyopathy and is characterized by unexplained myocardial hypertrophy (left ventricular wall thickness ≥15 mm in any myocardial segment) that cannot be attributed to conditions of increased load (i.e., arterial hypertension or valvular heart disease)[1] It is an inherited disease in which, in a large percentage (up to 60%) of cases, the responsible mutation is found in genes that encode cardiac sarcomere proteins. [2] Genetic testing is crucial in HCM management, with strong support from research studies and international guidelines.[1] The aim of the current study was to identify the potential genetic background in a population of 60 HCM patients.

Patients and Results

Sixty HCM patients (average age 46±12 years, 30 females) who are being followed-up at our hospital's Cardiomyopathy and Sudden Cardiac Death Prevention Department were studied. After providing signed informed consent, they underwent genetic testing using Next Generation Sequencing (NGS) technology. In 24 patients, no clinically significant genetic variants were identified. Twenty-one patients had mutations in the MYBPC3 gene, 10 in the MYH7 gene, 2 in the MYL2 gene in patients, 2 in the TPM1 gene, and 1 patient in the CSRP3 gene. Of the detected mutations, 20 were classified as pathogenic based on the American College of Molecular Biology and Genetics (ACMG) criteria, 8 were considered likely pathogenic, and 8 were categorized as variants of unknown significance (VUS) (Table 1). Subsequently, genetic testing of first- and second-degree relatives of patients with pathogenic/likely pathogenic mutations was conducted, and as a result, 15 carriers of these genes were identified. These carriers are currently phenotypically normal (electrocardiogram and echocardiogram without any HCM-specific findings). However, they have been placed under close follow-up, as they may develop the disease in the future.

Discussion

There is a clear role for genetic testing in HCM, in order to obtain diagnostic certainty for pro-

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bands. Our findings highlight the significant value of conducting genetic testing in HCM patients and to their relatives. In ~50% of cases, HCM appears to be inherited as a Mendelian genetic trait. In such cases, the inheritance is primarily autosomal dominant, i.e. with a 50% risk of transmission to offspring.[3] Until now, no definitive relationship has been established between individual sarcomere mutations and patterns of left ventricular hypertrophy in HCM.[4-6] Conversely, identifying pathogenic/likely pathogenic mutations in HCM patients allows for early identification of family members who carry these mutations, even if they are asymptomatic. This is important since asymptomatic gene carriers are placed on a regular follow-up, thus potentially preventing serious complications. On the other hand, for the family members that do not carry the disease-causing mutation, genetic testing can provide reassurance.

Finally, especially during the last decade, there is immense interest in the potential of genetic therapies in HCM. Many ongoing studies are focusing on the role of common genetic variation and the importance of cardiovascular risk factors in disease development and numerous trials are focused on gene replacement. These studies offer promise for more sophisticated disease models, and also allow the emergence of personalized approaches, including sarcomere modulation and genetic modification.[7]

The future is genetic testing and it is here. We just need to know when and how to use it.

	MYBPC3	MYH7	MYL2	TPM1	CSRP3	Total
Pathogenic	9	8	1	2	0	20
Likely pathogenic	6	1	1	0	0	8
VUS	6	1	0	0	1	8
Total	21	10	2	2	1	

Table 1. *Results of genetic testing in a cohort of 60 hypertrophic cardiomyopathy patients who are followed-up in our Clinic.*
VUS: variants of unknown significance, MYBPC3: myosin binding protein C3, MYH7 myosin heavy chain 7, MYL2: myosin light chain 2, TPM1: tropomyosin 1, CSRP3: cysteine and glycine rich protein 3

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CLINICAL ARTICLE

Electrocardiographic features in patients with hypertrophic cardiomyopathy - The Evangelismos experience

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Introduction

Hypertrophic cardiomyopathy (HCM) is one of the most common genetic cardiovascular diseases with an estimated prevalence of 0.2-0.5% in the general adult population.1 HCM is characterized by unexplained myocardial hypertrophy, meaning that it cannot be attributed to conditions of increased loading (i.e., arterial hypertension or valvular heart disease).1 It is inherited as an autosomal dominant trait in most cases, and the responsible mutation is found in genes that encode proteins of the cardiac sarcomere.[1] The standard 12-lead electrocardiogram (ECG) is considered a fundamental tool in the diagnosis and follow-up of HCM patients.[2,3]

Patients and Methods

We studied 95 HCM patients (average age 54 ± 5.8 years; 40 women, 55 men) who are followed-up at the Cardiomyopathy and Sudden Cardiac Death Prevention Clinic of Evangelismos Hospital, Athens, Greece. Apart from taking their medical history and conducting a thorough clinical examination, a standard 12-lead ECG and transthoracic echocardiography were performed during their first visit.

Results

Of these patients, 90 (94.7%) had an abnormal ECG, whereas in only 5 (5.3%) their ECG was normal *(Figure 1)*. 50 patients (52.6%) exhibited "non-specific" ST-T changes. Twenty-one patients (22.1%) displayed abnormal Q waves (amplitude ≥25% of the ensuing R-wave and/or duration ≥0.04 s) in the inferior and/or lateral leads, with associated repolarization abnormalities in these leads and positive electrocardiographic indicators of left ventricular hypertrophy *(Figure 2, panel A)*, while 10 patients (10.5%) had abnormal Q waves in the inferior and/or lateral leads, positive electrocardiographic indicators of left ventricular hypertrophy, but without repolarization abnormalities. Lastly, 9 patients (9.5%) exhibited giant negative T waves in the precordial leads *(Figure 2, panel B)*. In the aforementioned patients, the echocardiographic study confirmed the HCM diagnosis.

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Conclusion

The results presented in this research letter are consistent with international literature data.2,3 In patients with HCM, the 12-lead ECG is normal in a very small percentage of cases.2,3 It usually shows abnormalities at a very early stage of the disease's onset. This fact underscores the fundamental value of the standard 12-lead ECG in the diagnosis of HCM.

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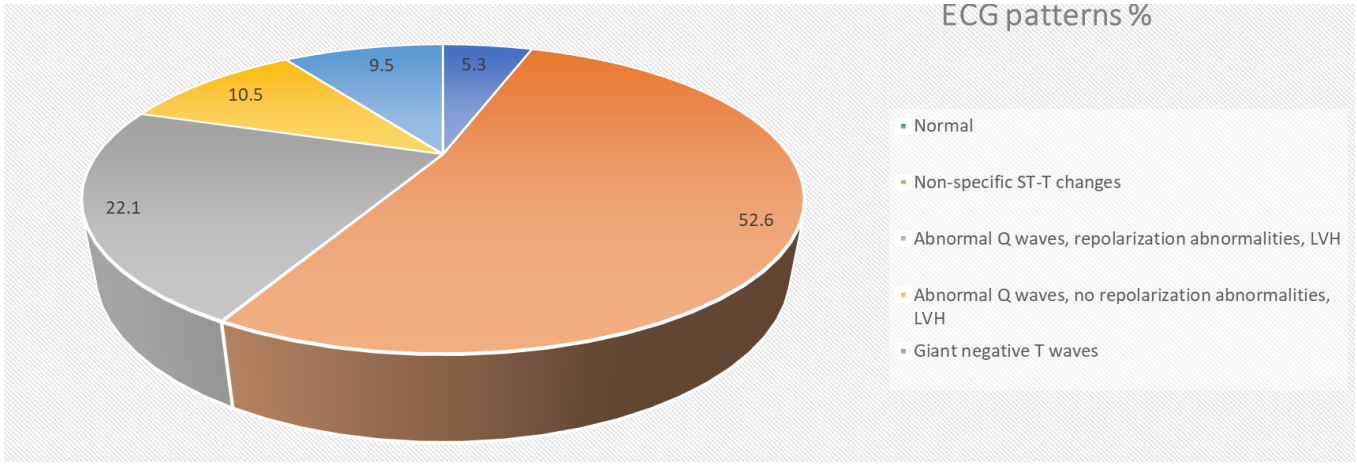

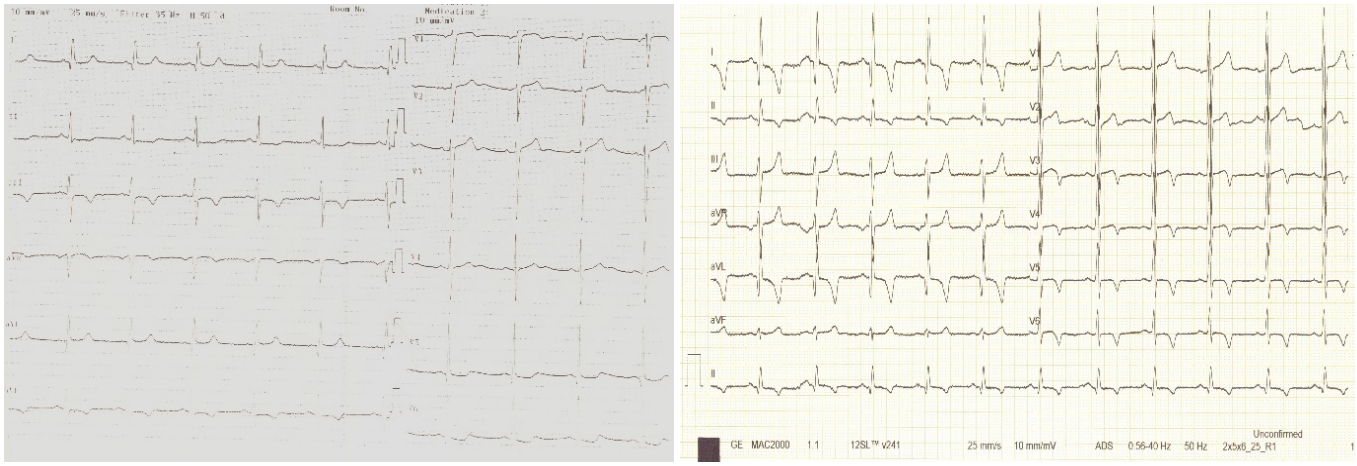

Figure 1. *Prevalence of ECG patterns in a cohort of 95 patients with HCM.*
Figure 2. *Characteristic ECG patterns suggestive of HCM*

CASE REPORT

Post-Cardiac Injury Syndrome and Pacemaker -associated Infection following Permanent Pacemaker Implantation

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Keywords: Permanent pacemaker, post-cardiac injure syndrome, pericardial effusion, pleural effusion, acute myopathy, pacemaker-related infection

Introduction

It is estimated that over one million cardiac pacemakers are implanted every year worldwide.1 In recent years, pacemaker (PM) technology has rapidly evolved and physicians' experience has significantly improved, making the implantation procedure much safer and the indications more extended. However, PM implantation is associated with the risk of potential complications, such as post-cardiac injury syndrome (PCIS) and PM-system infection.

PCIS refers to a heterogeneous group of conditions caused by autoimmune-mediated inflammation of the pericardium, epicardium, and myocardium. It includes three significant entities: post-myocardial infarction pericarditis, post-pericardiotomy syndrome (PPS) and post-traumatic pericarditis (either iatrogenic or not).2,3 PCIS is a rare complication following implantation of a permanent PM (PPM); it affects 1-2% of patients and the etiopathogenesis is not well defined.[4] It is mainly associated with screw-in leads positioned at the right atrium and the involved mechanisms may include minor lead perforation, inflammatory and autoimmune reactions.[5-9] The prognosis of the syndrome is in most cases benign and the first-line treatment of PCIS includes corticosteroids, non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine.[3]

Additionally, infections in PM-systems are serious complications, causing significant in-hospital morbidity, mortality and healthcare costs.[10,11] The lifetime risk of PM-associated infection is 1.19%.[12] Infection can involve either the generator pocket and/or the intracardiac portion of the device. Risk factors for cardiac implantable electronic device infection include comorbid conditions such as heart failure, diabetes mellitus and renal dysfunction, history of device infection, dual-chamber device, temporary pacing, device replacement, generator change, corticosteroid use, and oral anticoagulants.13 A successful treatment of a PM-related infection often requires the combination of complete removal of

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all parts of the PM system and the long-term administration of proper intravenous antibiotics.[13] We report a rare case of PCIS after dual-chamber PM implantation complicated with device-related infection, providing details of its evaluation and management.

Case presentation

A 72-year-old non-smoker female with a past medical history of paroxysmal atrial fibrillation (AF), hypothyroidism, arterial hypertension, dyslipidemia, and no prior history of autoimmune disease in her family was admitted to the Cardiac Intensive Care Unit due to syncope after oral loading with propafenone (300 mg) for conversion of recent-onset AF, as indicated by her cardiologist. The patient was taking bisoprolol (2.5 mg/day) and rivaroxaban (20 mg/day). The timeline of the patient's clinical course is shown in Table 1. At admission, the electrocardiogram (ECG) showed sinus rhythm with a ventricular rate of 40 beats per minute. Laboratory tests showed no relevant electrolyte disturbances, the C-reactive protein (CRP) was 0.84 mg/dl (reference range <0.30 mg/dl) and the level of thyroid stimulating hormone (TSH) was within normal range (TSH=1.751 μIU/ml, reference range 0.35-4.94 μIU/ml). The transthoracic echocardiography (TTE) study of admission revealed left ventricular ejection fraction of 60%, left atrial dilatation, severe aortic stenosis (aortic valve area 0.8 cm2), moderate tricuspid and mitral regurgitation, and no pericardial effusion. Telemetry monitoring of the patient revealed short episodes of AF and many symptomatic episodes of cardiac pauses >6 sec; hence a temporary PM was inserted via the right femoral vein, without immediate complications, followed by implantation of a dual chamber PPM two days later with two bipolar active fixation, screwing electrodes. Before the procedure, the patient received antibiotic prophylaxis (vancomycin). Two days after the implantation, the patient reported shortness of breath, weakness, and pleuritic chest pain. Cardiopulmonary auscultation revealed reduced heart sounds and absent breath sounds over the lower lung fields bilaterally with dullness to percussion. The PM scar had no inflammatory signs. The ECG showed sinus rhythm and laboratory tests revealed neutrophilic leukocytosis and elevated CRP levels. Based on these initial findings, the patient received empiric antibiotics for a possible pulmonary infectious process. Furthermore, the TTE showed large circumferential pericardial effusion (24 mm) with no signs of hemodynamic compromise and pleural effusion bilaterally (Video 1). A chest X-ray (CXR) showed cardiomegaly and bilateral pleural effusion, not present in the admission CXR. The PPM follow-up revealed no sign of PM dysfunction, sensing and pacing threshold being appropriate. The chest computed tomography (CT) did not show lead migration or protrusion and confirmed the presence of pericardial and bilateral pleural effusion *(Figure 1)*. The patient was hemodynamically stable and there were no clinical signs of cardiac tamponade, therefore pericardiocentesis was not performed. However, both pleural cavities were punctured, with drainage of total 2,000 ml of light-yellow fluid. The pleural fluid analysis showed sterile exudate (Table 2). Given all the above-mentioned findings, the patient was diagnosed with PCIS following the PM implantation and treated with ibuprofen (600 mg four times daily) and colchicine (0.5 mg twice daily). As there was no favorable clinical effect (pleural effusion increased, pericardial effusion remained the same), ibuprofen was discontinued on the third day and methylprednisolone

(16 mg twice daily) was introduced on the third day with a gradual resolution of pleural and pericardial effusions. Five days later, the patient developed gastrointestinal symptoms (nausea and diarrhea) and colchicine was reduced to 0.5 mg every other day with good tolerability. Furthermore, on day three of methylprednisolone treatment, she complained of myalgia and muscle weakness mainly in the arms and legs, symptoms consistent with acute myopathy caused by oral corticosteroids. Significant improvement was achieved 15 days later, after implementing daily physiotherapy, and gradually tapering off the dosage of methylprednisolone. The methylprednisolone dose was reduced after normalization of CRP and complete resolution of pericardial and pleural effusions. In addition, her course was complicated by many episodes of hematuria which were attributed to bladder diverticulum revealed by cystoscopy. Hematuria was successfully controlled with continuous bladder irrigation. The patient was discharged in a stable clinical condition on the 40th day of hospitalization receiving low dose of methylprednisolone (4 mg/day) and colchicine (0.5mg every other day). During her hospitalization, appropriate wound care of the site of PPM implantation was provided and the non-absorbable stiches were removed seven days following the implantation. Two months later, the patient was readmitted due to cardiac device infection. On initial examination, there was a non-healing surgical wound in the left upper chest at the site of PM implantation with erythema and yellowish purulent discharge with warmth and tenderness. She was afebrile, hemodynamically stable and the ECG showed AF. Relevant laboratory workup revealed no leukocytosis, but elevated CRP. Blood cultures for microorganisms were negative, including aerobic and anaerobic bacteria, and no clinical signs of infective endocarditis were observed. Cultures from the PM generator, the serum and wound revealed Staphylococcus epidermidis. TTE did not show any presence of lead-related vegetations. Empiric therapy with meropenem (6 gr/day), daptomycin (600mg/day) and gentamycin (240 mg/day) was initiated. Additionally, the heart team decided to remove the entire PM system (generator and leads) with debridement of the pocket. The day following the procedure, a hematoma at the surgical site was noted and operation for removing it was performed. Due to significant decrease in hemoglobin, replacement therapy with packed red blood cells and fresh frozen plasma was initiated. Rivaroxaban was temporary discontinued. Cultures of the electrodes revealed Staphylococcus auricularis and Staphylococcus hominis sensitive to the antibiotics she was already receiving and the same treatment was continued for the next four weeks. The patient has been doing well on follow-up examinations and instructions were given regarding the risk of relapse and to return if symptoms recur. In addition, the patient is scheduled for coronary angiography for preoperative detection of coronary artery disease, and transcatheter aortic valve implantation (TAVI). The implantation of a new PPM remains under consideration.

Discussion

While PCIS has been found to be a relatively common complication of open-heart surgery with an incidence of 10-40%, its incidence following PPM implantation remains quite low with the estimated risk to be between 1% and 2%.[4,14-16] A recent study by Fibley et al. showed an even lower incidence of 0.38%, finding which may be explained by the use of more modern lead designs.[17] Although the precise

pathomechanism of PCIS after PPM implantation remains not clear, an immune-mediated inflammatory reaction triggered by slightly protruding electrodes which irritate directly the pericardium with or without bleeding has been proposed as a possible mechanism.[17-19] Hence, the syndrome is more likely to develop in patients with screw-in leads positioned at the thinner right atrial wall where micro-perforations may occur.[4-9,17]

The clinical manifestations of PCIS are non-specific, making the diagnosis of the syndrome challenging, and include shortness of breath, fever, chest pain, pericardial or/and pleural effusion and elevated inflammatory markers. PCIS must be differentiated from simple pericardial or pleural effusion often observed after cardiac surgery, where excessive inflammatory activity is not present. In addition, clinical entities such as idiopathic or infectious pericarditis and delayed perforation must be ruled out. The diagnosis of PCIS, based on the proposed criteria from 2015 European Society of Cardiology (ESC) guidelines, requires a history of cardiac injury and at least 2 of the following 5 criteria to be fulfilled: (i) fever without alternative causes, (ii) pericarditic or pleuritic chest pain, (iii) pericardial or pleural rubs, (iv) evidence of pericardial effusion and/or (v) pleural effusion with elevated CRP. Our patient underwent PPM implantation with screw-in leads (cardiac injury) and fulfilled three of the above five criteria (pleuritic chest pain, pericardial effusion and pleural effusion with elevated CRP); hence, the diagnosis of PCIS was established.[3] Female gender, advanced age, and use of screw-in leads are independent risk factors for the development of pericarditis after PPM insertion, factors which our patient had.20 Symptoms mainly occur within two weeks after implantation, with a median onset of 7 days.[17] In our case, the onset of symptoms was two days after the procedure.

First-line treatment of post-PM PCIS is no different from other forms of PCIS and includes aspirin, NSAIDs, corticosteroids and colchicine. If initiated early, most patients will have a good clinical response and the need for invasive procedures, such as pericardiocentesis or pericardial window, will be minimized. Hence, delayed diagnosis and medical treatment may prolong in-hospital stay and healthcare costs.[10] In our case study, the patient responded well to the oral medical therapy which included methylprednisolone and colchicine with gradual complete resolution of pericardial and pleural effusions. Colchicine was administrated simultaneously along with NSAIDs and corticosteroid, since it has been shown to improve the response to medical therapy and lower the recurrence rate.[21-23] Our patient suffered from diarrhea, a well-known side effect of colchicine, which was treated by reducing the initial dosage.[24] Furthermore, after the administration of oral methylprednisolone, the patient suffered from symptoms, such as diffuse myalgia and muscle weakness especially at the lower limps. These symptoms posed a diagnostic dilemma whether the patient was weaker due to PCIS exacerbation or due to the steroids. We concluded that the cause was most likely steroid-induced myopathy as the patient felt better when steroids were being tapered. On the other hand, a truly acute myopathy after oral corticosteroid administration is very rare and remains the subject of very few, single case reports, while it typically occurs after high intravenous dosage of steroids.[25-27]A number of laboratory investigations, such as AST, ALT, CPK, and urine myoglobin may aid

in the diagnosis of acute steroid myopathy. In our patient CPK was not elevated, which is a usual finding in many reported cases of steroid-induced myopathies.[28]

Additionally, our patient suffered from pocket PM infection. The risk factors for these infections include dual-chamber device, temporary pacing, device replacement, generator change and use of corticosteroids and oral anticoagulants, factors which our patient had, putting her at high relative risk for cardiac implantable electronic device (CIED) infection.[13] Samples for bacterial cultures taken from the pocket and trauma revealed Staphylococcus epidermidis, a common microorganism for CIED infections.[10] Furthermore, cultures from the ventricular lead revealed Staphylococcus auricularis and Staphylococcus hominis. All pairs of blood cultures taken before the antibiotic therapy were negative, our patient did not have clinical manifestations suggestive of sepsis and her procalcitonin was not elevated. Hence, Staphylococcus auricularis and Staphylococcus hominis were considered to be due to contamination of the leads during the PM implantation procedure. It is known that cardiac electrophysiological devices can be colonized by bacteria without clinical signs of infection.[29] The above-mentioned microorganisms were sensitive to the empiric antibiotic therapy she was started on, which was specific for Staphylococcus epidermidis, and at the end they did not affect neither her treatment nor her prognosis.

The day after the complete removal of the PM system, a pocket hematoma was noted and operation for removing it was performed, a complication that worsened her clinical condition. In addition, the need for reimplantation of the PM was called into question by the heart team, because her syncope and symptomatic episodes of cardiac pauses >6 sec appeared after taking antiarrhythmic drugs and the risk of relapsing infection was considered high. Furthermore, the patient refused to get a new device. It should be kept in mind that around one third to one half of patients, who had their PM completely removed due to infection, may not require a reimplantation.[30]

Conclusion

In the last decades, there has been an increase in the number of PPM implantations, and because of that we have witnessed increased rates of PM-related complications, such as PCIS and infections. Hence, it is extremely important to carefully assess conditions who lack any beneficial effect from cardiac pacing before the implantation of a PPM.

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Time	Event
Admission	Episode of syncope, severe aortic stenosis.
Day 1	Monitoring revealed short episodes of atrial fibrillation and sinus pauses. Temporary pacemaker implanted.
Day 3	Permanent pacemaker implantation.
Day 5	Shortness of breath, pericardial and pleural effusions.
Days 5-8	Ibuprofen and colchicine without significant clinical effect
Day 8	Methylprednisolone and colchicine with gradual resorption of pericardial and pleural effusions.
Day 10	Oral steroid-induced myopathy.
Day 13	Nausea and diarrhea. Colchicine dose reduction.
Day 19	Gradual reduction of methylprednisolone dose.
Day 40	Discharge.
Day 93	Re-admitted. Pocket infection. Antibiotic therapy.
Day 94	Removal of the entire pacemaker system.
Day 95	Hematoma at the surgical site.
Day 135	Discharge in good clinical condition.

Table 1. *Timeline of the patient's clinical course*

Analysis of pleural fluid	
Appearance	Yellow
Microbiology	Sterile
Nucleated count cell (per cubic millimeter)	1,360
Neutrophils (%)	70.6
Lymphocytes (%)	19.9
Total protein (g/dl)	1.7
Albumin (g/dl)	1.2
LDH (IU/l)	386
Fluid to serum LDH ratio	2.0
Fluid to serum Albumin ratio	0.4
Cytology report	Negative for malignant cells

Table 2. *Pleural fluid analysis and culture*

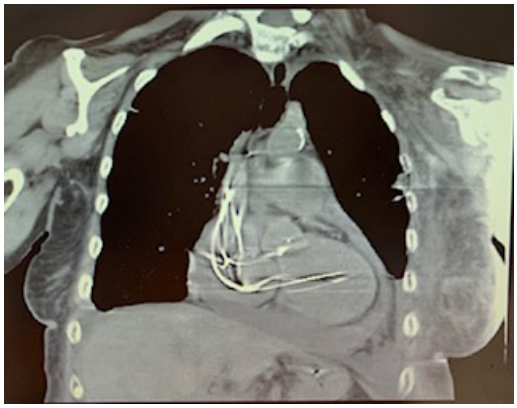

Fig 1. *Bilateral pleural effusion*

CASE REPORT

Takotsubo Syndrome in patient with Hypertrophic Obstructive Cardiomyopathy (HOCM)

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Introduction:

Takotsubo syndrome (TTS), also referred to as stress-induced cardiomyopathy, is a transient and reversible form of acute heart failure that clinically and electrocardiographically mimics acute myocardial infarction. The most common morphological pattern is characterized by transient apical ballooning, manifested as akinesia, hypokinesia, or dyskinesia of the apical segments of the left ventricle (LV), with preserved or hyperkinetic function of the basal segments.[1] Although the precise pathophysiology of TTS is not completely elucidated, current evidence implicates a multifactorial mechanism involving exaggerated sympathetic activation, coronary microvascular dysfunction, epicardial coronary vasospasm, and direct catecholamine-mediated myocardial toxicity. These processes are typically triggered by acute physical or emotional stressors, certain neurologic disorders, or catecholamine-secreting tumors such as pheochromocytoma.[1]

In patients with preexisting hypertrophic cardiomyopathy (HCM), a distinct variant of TTS has been reported in association with acute dynamic left ventricular outflow tract obstruction (LVOTO). In such cases, obstruction-induced hemodynamic compromise may precipitate the development of TTS, particularly in the presence of a marked intraventricular pressure gradient. [3,8]

Case presentation:

A 66-year-old male patient with a known history of hypertrophic obstructive cardiomyopathy (HOCM) and preserved left ventricular (LV) systolic function presented to our hospital with complaints of vomiting, palpitations and presyncope. On examination, he was found to be hemodynamically unstable. Shortly thereafter, he developed sustained monomorphic ventricular tachycardia, which was promptly terminated with electrical cardioversion.

Initial electrocardiogram (ECG) demonstrated features consistent with left ventricular hypertrophy (LVH) and associated repolarization abnormalities (strain pattern). Bedside transthoracic echocardiography revealed akinesis of the apical and apical-septal segments, with compensatory hypercontractility of the basal LV segments. The estimated LV ejection fraction was approximately 35%. Serum troponin I was

CITATION: Efthymiou P., et al, Takotsubo Syndrome in patient with Hypertrophic Obstructive Cardiomyopathy (HOCM), CyJCardMed 2025, 3, 1, 30-34.

elevated at 3.52 ng/mL.

Given the clinical and echocardiographic findings, the patient underwent urgent coronary angiography, which identified a moderate, stable stenosis in the mid-segment of the left anterior descending (LAD) artery *(figure 1)*. Physiological assessment using instantaneous wave-free ratio (iFR) yielded borderline values of 0.89–0.90, and percutaneous coronary intervention (PCI) was performed. Left ventriculography revealed classic "apical ballooning" consistent with takotsubo syndrome *(figure 2)*. Hemodynamic measurement using end-hole catheter revealed significant pullback pressure gradient at the level of LVOT (37mmHg), consistent with dynamic obstruction *(figure 3)*.

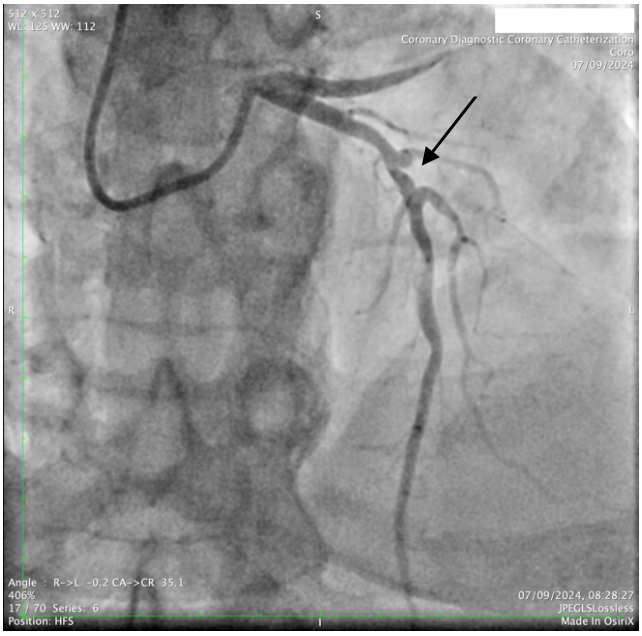

Figure 1: *Stable mid LAD stenosis (black arrow)*

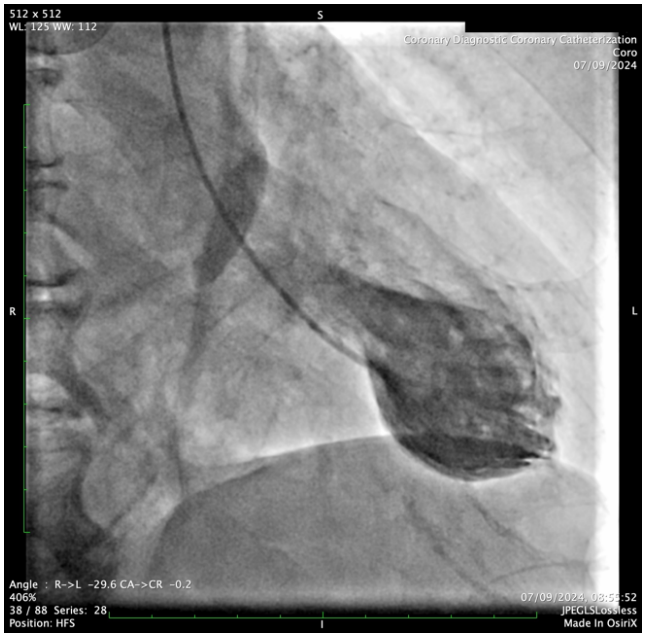

Figure 2: *Apical ballooning in left ventriculography*

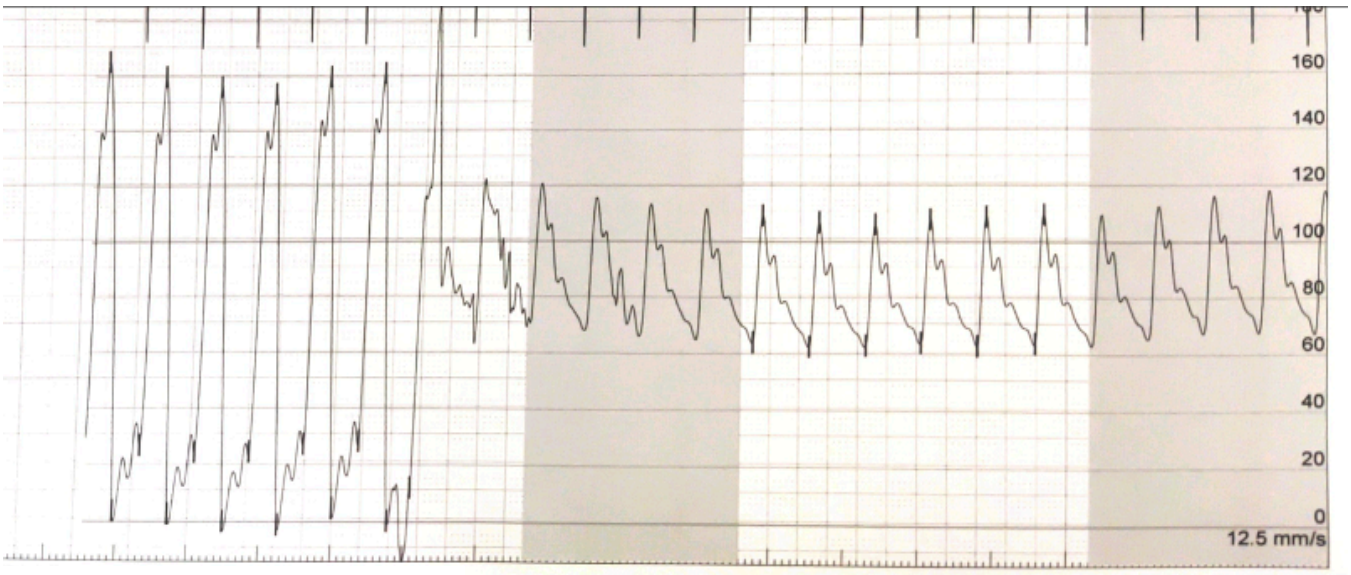

Figure 3: *high pressure gradient at the level of LVOT*

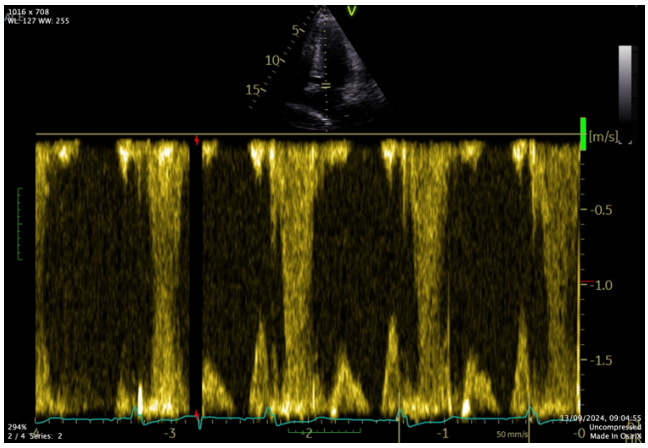

Figure 4: *Echocardiography post normalization function showing high velocities in LVOT*

Repeat echocardiography on the following day showed normalization of LV systolic function, focal hypertrophy of the interventricular septum (IVS) with septal thickness measuring 16 mm and LVOT obstruction *(figure 4).* Cardiac magnetic resonance imaging (MRI) was performed for further diagnostic clarification. It confirmed preserved global LV contractility, asymmetric hypertrophy involving the basal and mid-IVS and turbulence flow in LVOT due to systolic anterior motion (SAM) of the mitral valve leading to obstruction. Additionally, myocardial oedema was present in all apical segments in T2 weighted black blood images, and mild mid-wall late gadolinium enhancement was observed in the same regions (figure 5). These findings were consistent with acute non ischemic myocardial injury pattern in the setting of HOCM, supporting a diagnosis of takotsubo syndrome.

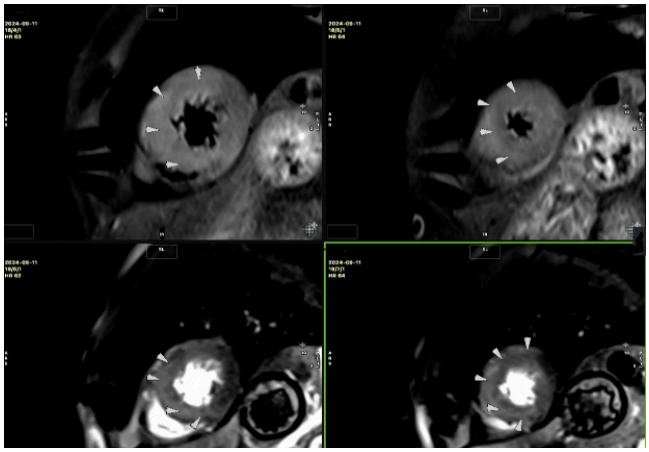

Figure 5: *CMR showing myocardial oedema in T2 weighted images (first row) and mid wall LGE (second row)*

The patient was managed with intravenous fluids and vasoconstrictive agents, to which he responded positively. An implantable cardioverter-defibrillator (ICD) was placed prior to discharge for secondary prevention of sudden cardiac death.

Discussion:

This case highlights a rare and diagnostically complex overlap between hypertrophic obstructive cardiomyopathy and Takotsubo syndrome, underscoring the intricate interplay between mechanical and neurohumoral factors.

In our patient, the acute presentation with presyncope, vomiting, and hemodynamic instability progressed rapidly to sustained monomorphic ventricular tachycardia (VT), necessitating electrical cardioversion. Transthoracic echocardiography revealed hallmark features of TTS, including akinesis of the apical and apical-septal segments with basal hypercontractility, alongside a significant drop in estimated LV ejection fraction to 35%. Coronary angiography revealed significant yet but stable coronary artery disease, with a borderline mid-LAD lesion treated with percutaneous coronary intervention. Left ventriculography confirmed apical ballooning, while simultaneous catheterization revealed a dynamic LVOT gradient of 37 mmHg, supporting the diagnosis of HOCM-related obstruction. According to the latest and revised diagnostic criteria proposed by the European Society of

Cardiology (interTAK criteria), the presence of significant coronary artery disease in coronary angiography does not exclude the presence of TTS. [1,6]

CMR has emerged as a valuable adjunctive modality in the diagnosis and management of Takotsubo syndrome, offering detailed assessment of myocardial structure, function, and tissue characterization. Myocardial edema is the main finding and represents the inflammation secondary to transient ischemia or increased myocardial wall stress. It is visualised with several techniques such as "T2-weighted block blood imaging" , native T1 mapping or T2 mapping. The distribution of the edema and the fast resolving reinforce the concept of TTS as a transient ischemic insult rather than infarction.[1,9]

The pathophysiological overlap between HOCM and TTS remains incompletely understood but may be conceptualized along two primary mechanisms. First, catecholamine excess in response to stressors can precipitate apical dysfunction in predisposed individuals, including those with HOCM. Second, the dynamic obstruction inherent to HOCM can itself serve as a mechanical trigger for TTS, particularly when augmented by dehydration, hypovolemia, or positive inotropic states. Our patient likely represents the latter scenario, wherein transient LVOT obstruction led to afterload mismatch, subendocardial ischemia, and ultimately the classic TTS phenotype.

Management in such cases must be carefully tailored. Beta-blockers are standard in treatment and phenylephrine is used to manage hypotension. Diuretics should be used cautiously and only to relieve pulmonary edema, as they can reduce preload and worsen intraventricular gradients. IV fluids may help by increasing preload and reducing obstruction. Positive inotropes, typically used in cardiogenic shock, should be avoided in LV ballooning with LVOTO, as they exacerbate systolic anterior motion (SAM) and obstruction. Catecholamines are particularly risky in TTS-related shock and may worsen clinical outcomes. As a safer, catecholamine-sparing option, nonadrenergic inotropes such as the calcium sensitizer levosimendan may be considered. [2,3,5,10] For refractory shock with LVOTO, mechanical circulatory support may be considered. The application of a percutaneous LV assist device (Impella) can bypass LVOT ejecting blood directly into aorta but may reduce preload. Intra-aortic balloon pumps (IABP) are contraindicated as they lower afterload and may worsen the obstruction. Venoarterial ECMO may be preferred in severe cases with end-organ hypoperfusion.[3,7,8]

Conclusion

This case emphasize an underrecognized but clinically significant variant of Takotsubo syndrome triggered by dynamic LVOT obstruction in a patient with hypertrophic obstructive cardiomyopathy. Recognition of this interplay is critical for accurate diagnosis, appropriate hemodynamic management, and prevention of adverse outcomes. As awareness of this overlap increases, clinicians should maintain a high index of suspicion in similar presentations and incorporate detailed assessment into the diagnostic workup and treatment.

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INTERESTING CASES

Investigation of cardiac conduction disorders in two young patients; the role of cardiogenetics

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Abstract

Objective: This report aims to highlight the diagnostic approach and role of cardiogenetics in managing cardiac conduction disorders in young patients.

Methods and Results: We present two cases involving young adults with cardiac conduction abnormalities.

Case 1: A 38-year-old male presented with fatigue and reduced exercise tolerance. Initial investigations, including ECG, Holter monitoring, and blood tests, revealed sinus rhythm with episodes of 2:1 and 3:1 atrioventricular block. A cardiac MRI indicated subepicardial scarring, suggesting past myocarditis. Genetic testing identified variants of uncertain significance in four genes. Despite recommendations, the patient declined pacemaker implantation and remains under regular follow-up.

Case 2: A 32-year-old female reported dizziness and exercise intolerance. Family history included sudden cardiac death in her uncle. ECG and Holter monitoring indicated normal sinus rhythm with intermittent 2:1 atrioventricular block. Cardiac MRI showed mid-wall septal scarring. Genetic testing revealed an LMNA mutation, leading to a diagnosis of laminopathy. An ICD was implanted due to the risk of malignant arrhythmias.

Conclusion: The cases underscore the importance of genetic testing in diagnosing and managing cardiac conduction disorders in young patients. Genetic insights can refine diagnosis, inform treatment strategies, and guide familial risk assessment, underscoring the evolving role of cardiogenetics in personalized medicine.

Keywords: Cardiac conduction disorders, cardiogenetics, genetic testing, laminopathy, pacemaker, ICD, myocarditis, AV block.

Introduction

Cardiac conduction disorders in young patients pose significant diagnostic and therapeutic challenges, often requiring a nuanced approach that incorporates advanced genetic insights.

CITATION: Vrettos Ap., et al, Investigation of cardiac conduction disorders in two young patients; the role of cardiogenetics, CyJCardMed 2025, 3, 1, 35-39.

This case series aims to illustrate the diagnostic strategies and the critical role of cardiogenetics in managing these complex conditions. By presenting two cases of young adults with distinct cardiac conduction abnormalities, we highlight how genetic testing can refine diagnoses, influence treatment decisions, and guide familial risk assessments, ultimately enhancing personalized medical care in cardiology.

Case 1

This case report presents the case of a 38-year-old male patient who was referred to our department for the investigation of cardiac conduction disorder on resting electrocardiogram (ECG) and Holter monitoring *(Fig. 1a)*. He complained of fatigue and reduced exercise tolerance for 1 year. There was no family history of sudden cardiac death, arrhythmias, syncopal episodes or known cardiomyopathy. A full blood count, U/Es, and autoimmune screen were unremarkable. A Holter monitor showed sinus rhythm with right bundle branch block (RBBB), chronotropic incompetence with a minimum heart rate (HR) of 39 beats per minute (bpm), and periods of 2:1 and 3:1 atrioventricular block (AVB). His resting ECG on his first visit in our department showed sinus rhythm with 1st degree AVB and RBBB. A transthoracic echocardiographic (TTE) study did not show any significant abnormalities. Coronary angiography did not reveal any significant stenosis. A cardiac magnetic resonance (CMR) imaging showed subepicardial scarring of the lateral wall, possibly suggestive of previous episode of myocarditis (Fig. 1b). He underwent PET-CT scanning which did not reveal any pathological uptake in the myocardium of the left ventricle, ruling out active cardiac sarcoid. Genetic testing revealed variants of uncertain significance (VUS) in 4 genes (ABL1, COL5A1, NEXN and TTN). He then underwent an electrophysiology (EP) study where intravenous administration of isoproterenol did not increase the HR, indicating poor chronotropic response, and the recommendation was to implant a permanent dual-chamber cardiac pacemaker. The patient did not consent for the procedure and has remained under regular annual follow-up in our department.

Case 2

A 32-year-old female presented with episodes of dizziness and exercise intolerance. She had no relevant past medical history. Her family history was notable for sudden cardiac death in her paternal uncle at age 49. A full blood count, U/Es, and autoimmune screen were unremarkable. The ECG revealed normal sinus rhythm with 58 bpm. A Holter monitor showed sinus rhythm and periods of 2:1 AVB *(Fig. 2a)*. Further evaluation with TTE showed normal left ventricular function without structural abnormalities. A CMR scan showed normal biventricular size and function with small amount of left ventricular mid-wall septal scarring *(Fig. 2b)*. A PET-CT scan did not reveal any pathological uptake in the myocardium of the left ventricle. Genetic testing identified a heterozygous mutation in the LMNA gene (NM_005572.3:c.148C>T p.(Arg50Cys)), confirming a diagnosis of laminopathy. Given the risk of progression to complete heart block, risk of malignant ventricular arrhythmias and sudden cardiac death, the patient underwent cardioverter-defibrillator (ICD) implantation. She has remained under regular annual follow-up in our department.

Causes of high-degree atrioventricular block in people of various ages

In older adults, AV block is often due to age-related fibrosis of the conduction system, ischemic

heart disease, and degenerative conditions such as Lev's or Lenègre's disease. In contrast, high-degree AV block in young people can be caused by genetic mutations (e.g. SCN5A, LMNA genes), congenital heart diseases, myocarditis, and autoimmune conditions such as lupus and sarcoidosis. Understanding the age-specific etiologies is crucial for accurate diagnosis, risk stratification and targeted management.[1-4] Family history of pacemakers often indicates an underlying genetic predisposition to conduction system diseases. Even when an obvious cause cannot be identified after the initial work-up, these patients should remain under regular follow-up, as many of the currently unknown genetic or other conditions may declare themselves over time, some of which may necessitate modifications to current treatment (e.g. upgrade of implanted device to ICD if a highly arrhythmogenic condition becomes apparent such as cardiac sarcoid).[5,6]

The role of cardiogenetics

Cardiogenetics has profoundly transformed the diagnosis and management of conduction disease in young people. Recent research has identified several key genes associated with cardiac conduction disorders. Mutations in SCN5A, DES, HCN4, and LMNA genes have been implicated in various pathologies affecting cardiac rhythm.[7] For instance, SCN5A mutations are linked to Brugada syndrome and progressive cardiac conduction disease, highlighting the critical role of ion channels in maintaining normal cardiac electrical activity. By pinpointing the genetic basis of these disorders, cardiogenetics has enhanced diagnostic accuracy beyond traditional methods such as ECG and Holter monitoring, which often fail to reveal the underlying etiology.[8-10]

The ability to identify genetic mutations has enabled the development of personalized treatment strategies. For patients with SCN5A mutations, for example, avoidance of certain drugs and the aggressive treatment of fever is generally recommended. Individuals with LMNA mutations may require early implantation of ICDs. This targeted approach ensures that patients receive the most appropriate and effective treatment based on their unique genetic profile.[5,8]

Genetic testing also plays a crucial role in risk stratification and preventive care. By identifying high-risk mutations, clinicians can better predict the likelihood of adverse events and implement timely interventions. Family screening becomes particularly important in this context, as it allows for the detection of asymptomatic carriers who might benefit from early monitoring and intervention.[8] Lifestyle modifications and regular follow-up can be recommended to individuals with predisposing genetic mutations, potentially delaying disease progression and improving long-term outcomes.

Despite these advancements, the field of cardiogenetics faces several challenges. The interpretation of VUS remains a significant hurdle, as the clinical implications of these genetic alterations are not well-defined.[11] Continued research and the expansion of genetic databases are essential to better understand these variants and their impact on cardiac health. Moreover, the integration of genetic testing into routine clinical practice requires a multidisciplinary approach involving cardiologists, geneticists, and genetic counselors. Comprehensive education and training for healthcare providers are necessary to ensure accurate interpretation and effective communication of genetic results to patients and their families.[12]

In conclusion, the cases underscore the importance of genetic testing in diagnosing and managing cardiac conduction disorders in young patients. Cardiogenetics have revolutionized the

management of conduction disease in young people by providing a deeper understanding of the genetic basis of these conditions. The ability to identify specific mutations has led to personalized treatment strategies, improved risk stratification, and preventive measures. As research continues to evolve, the integration of genetic testing into clinical practice is expected to become more widespread, ultimately enhancing patient outcomes and paving the way for precision medicine in cardiology.

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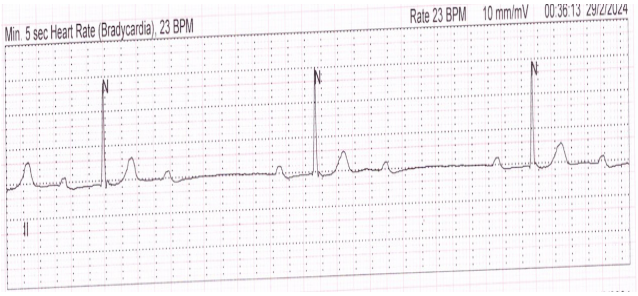

Fig 1. *Episodes of 2:1 AV Block*

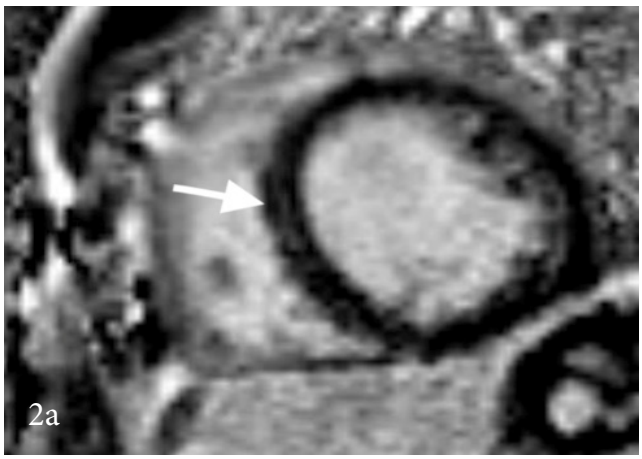

Fig 2a. *CMR scan. Subepicardial delayed gadolinium enhancement of the lateral wall at mid-left ventricle*

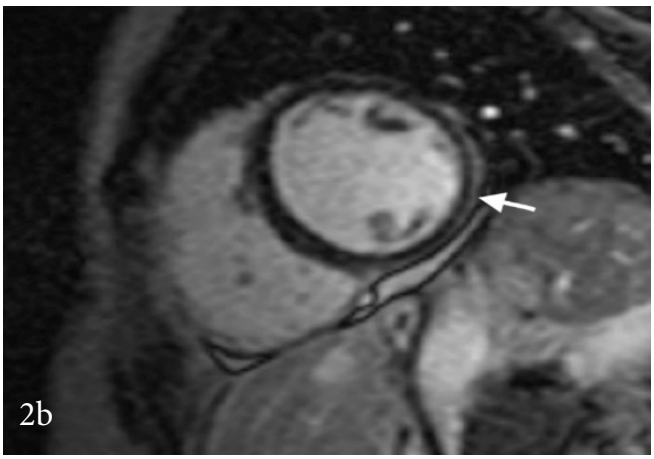

Fig 2b. *CMR scan. Mild linear mid-wall scarring of the interventricular septum at mid-left ventricular level*

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.

Trainees are eligible to sit the exam in their third year of their specialty training.

Trainees who do not achieve the required standard to pass the exam will be able to continue their training and resit the exam in the following year.

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.

Objectives of the mock exam are to provide an overview of the exam, the format of the questions and to start testing candidates' knowledge on the different chapters of the core curriculum.

The following modules are part of the EECC.

Module 1. Imaging
Module 2. Coronary artery disease
Module 3. Valvular heart disease
Module 4. Rhythm disorders
Module 5. Heart Failure
Module 6. Acute Cardiovascular Care
Module 7. Prevention, rehabilitation, sport
Module 8. Cardiac patients in other settings

A Certificate is issued to the candidates, who pass the Exam, by the Cyprus Society of Cardiology in collaboration with the ESC and the UEMS.

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The manuscripts should be prepared within the following limits.

SECTIONS	WORDS IN ABSTRACT (up to)	WORDS IN MAIN TEXT (up to)	REFERENCES (up to)	FIGURES OR TABLES (up to)
Original and review articles	250	3000	50	8
Case Reports	250	1000	10	2
Editorial & Letter to the Editor	–	1000	10	1
Images in Cardiology	–	500	5	2

Articles Should Be Organized Into Following Sections

Clinical and Experimental Articles: Title page, Abstract and key words. Introduction, Methods, Results, Discussion, Conclusions, Acknowledgements, References, Figures, Tables.

Review Articles: Title page, Abstract and key words. Subheadings in main text, Acknowledgements, References, Figures, Tables.

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(2) Abstract and key words. The abstract should be comprehensive but concise consisting of no more than 250 words and should be structured into four paragraphs in clinical/experimental articles: Introduction, Methods, Results, Discussion, Conclusions. In review articles, it should be undivided. The abstract should be followed by a list of 5-7 carefully chosen keywords. Only common abbreviations should be used in the abstract.

(3) Introduction. Introduction should include state of knowledge up-to-date and the aim of the study.

(4) Methods. Methods should describe the applied methods for the study, the participants and the statistical analysis. The source of the various materials used in the study should be given, where possible.

(5) Results. Results should be presented in a logical and simple fashion. Figures and tables should be included to make the manuscript more readable.

(6) Discussion. The results described above should be discussed in the light of any previous available studies in the same field. There is no need to repeat the results in this section.

(7) Conclusions. Conclusions of the study based on the aims and the results should be presented briefly and clearly in one paragraph.

(8) References. Authors must ensure that all references are cited accurately and those in the main text body are also included in the list of references and vice versa. References should list all the bibliographical sources cited in the text, in the order of their appearance. Citations in the text should be put in square brackets. Standard abbreviations should be used for journal names (according to Index Medicus). All references should be compiled at the end of the paper in the Vancouver style (Reference number, first 3 authors then "et al", title of the article, journal title (as it appears in Index Medicus), year of publication, volume and/or issue, number, first and last page separated by a dash. Reference to a specific chapter of a book, should include the name of the author, the chapter title, the editor, the place and year of publication and the first and last page.

(9) Acknowledgements. The authors may acknowledge any personal support for the work and any support in in the form of grants.

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