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16th CHALLENGES in CARDIOLOGY

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POSTERS

16TH CHALLENGES IN CARDIOLOGY

CLINICAL CASES

A rare pathway of spread: pulmonary adenocarcinoma extending into the left atrium

André Manuel Martins; Rita Martins; Mónica Amado; Joana Pereira; Adriana Vazão; Carolina Esteves; Ricardo Cordeiro; Mariana Saraiva

HOSPITAL DE SANTO ANDRÉ

An 84-year-old man presented with pleuritic chest pain, dyspnoea, and hemoptoic sputum. Chest CT revealed a perihilar pulmonary mass in the right lower lobe measuring 8.5×5.2×3.8 cm (Figure 1b), along with multiple bilateral nodules, some cavitated and exhibiting a feeding vessel sign. Bronchoscopy, including endobronchial biopsy, bronchoalveolar lavage, and bronchial aspirate, yielded inconclusive results. CT-guided biopsy confirmed a non-small cell lung carcinoma consistent with primary pulmonary adenocarcinoma. In light of new-onset fever and elevated inflammatory markers, transthoracic echocardiography was performed to exclude infective endocarditis. The exam revealed a left atrial mass (Figure 1a), further characterized by transesophageal echocardiography as a 2.8×3.4 cm heterogeneous lesion originating from the right inferior pulmonary vein (Figure 2). Contrast-enhanced echocardiography demonstrated perfusion patterns comparable to adjacent myocardium (Figure 3), findings consistent with an intracardiac tumour. Altogether, findings supported cardiac invasion by the pulmonary malignancy. Despite supportive measures, the patient experienced rapid clinical deterioration and died during hospitalisation.

Recurrent coronary in-stent restenosis during peptide receptor radionuclide therapy: the role of cardio-oncology in high-risk cardiovascular patients

Leandro Augusto Silva1; Raquel G. Martins[1]; Rita Garcia[1]; Sara Melo[1]; Sofia Oliveira[1]; Tomás Carlos[2]; Elisabete Jorge[2]; Catarina Neves[1]

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[2] UNIDADE LOCAL DE SAÚDE DE COIMBRA

Introduction

Cardio-oncology aims to minimize unnecessary interruptions to cancer therapy through multidisciplinary, individualized decision-making. Neuroendocrine tumours (NET) grow slowly, and heart disease may be the greatest limiting factor of overall prognosis. Managing coronary disease in cancer patients requires tailored strategies.

Clinical case

A 75-year-old male with metastatic VIP-secreting pancreatic NET, diagnosed at 67 years old, underwent pancreaticoduodenectomy and later developed hepatic metastases treated with lanreotide and thermoablation. His cardiac history included significant left main coronary artery (LMCA) and 3-vessel disease treated with coronary artery bypass grafting (CABG) using the left internal mammary artery (LIMA) to left anterior descending artery (LAD) and saphenous vein graft to obtuse marginal, as well as drug-eluting stent (DES) implantation in LMCA-left circumflex artery (LCx) and proximal LCx territories. He was also submitted to biologic aortic valve replacement and pacemaker implantation.

Five years later, peptide receptor radionuclide therapy (PRRT) with [^{177}Lu]Lu-DOTA-TATE was proposed. Cardio-oncology evaluation found no contraindications: troponin and NT-proBNP levels were normal, and transthoracic echocardiography showed preserved systolic function. He was maintained on acetylsalicylic acid (ASA) and clopidogrel due to complex coronary anatomy.

Two months after cycle 1, he presented with palpitations, vomiting, and elevated troponin levels. Coronary angiography revealed in-stent restenosis at LMCA-LCx (treated with a new DES) and proximal LCx (balloon angioplasty). Antiplatelet therapy was escalated to ASA/ticagrelor and PRRT suspended.

After optimisation, cycle 2 was administered. One month later, chest pain prompted repeat angiography, revealing recurrent restenosis at both sites, treated with drug-eluting balloons (DEB). Therapy was switched to ASA/prasugrel. Following multidisciplinary review, PRRT was permanently discontinued; and no further angina has occurred. Disease remains controlled with lanreotide.

Discussion

This case highlights cardio-oncology's role in enabling antineoplastic therapy in high-risk patients and supports cardiovascular monitoring before cardiotoxic treatments. NET's prolonged survival justifies aggressive cardiac intervention. Metastatic disease may promote a pro-thrombotic state, accelerating restenosis, while bleeding risk demands individualized antiplatelet strategies. PRRT discontinuation due to recurrent ischemia illustrates the balance between cancer treatment and cardiac stability. Although no solid evidence links PRRT to stent restenosis, the temporal relationship justified discontinuation.

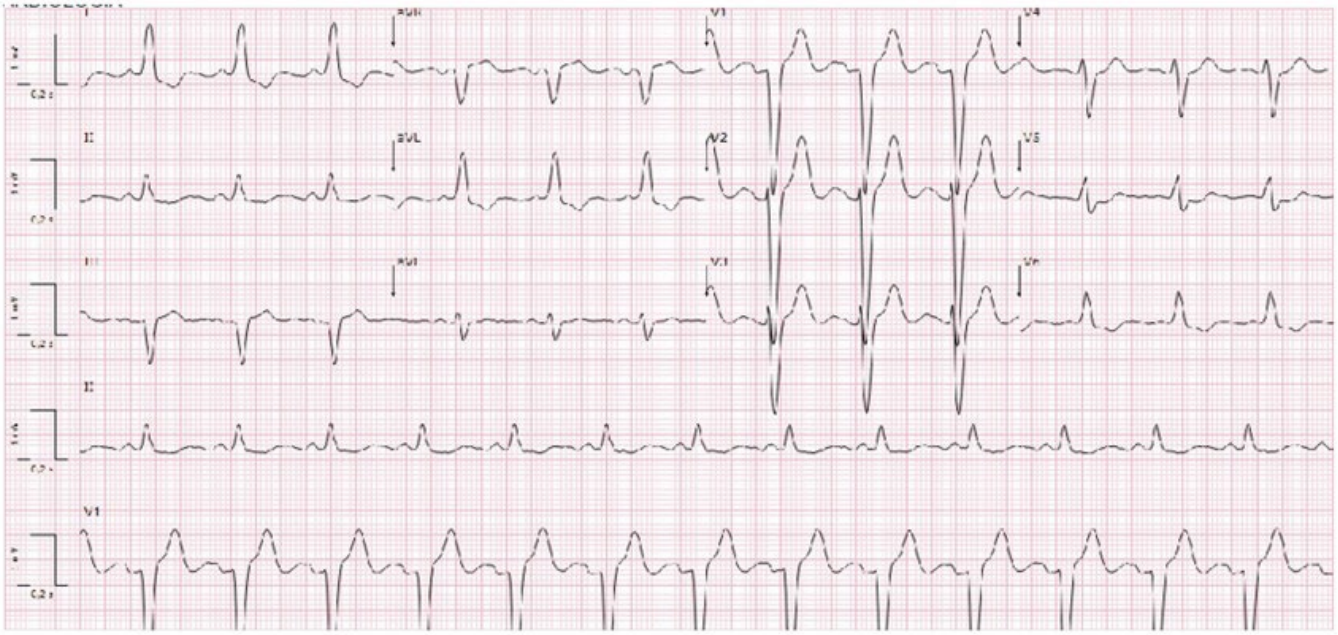

Figure 1 - Electrocardiography demonstrating normal sinus rhythm with previously existing left bundle branch block.

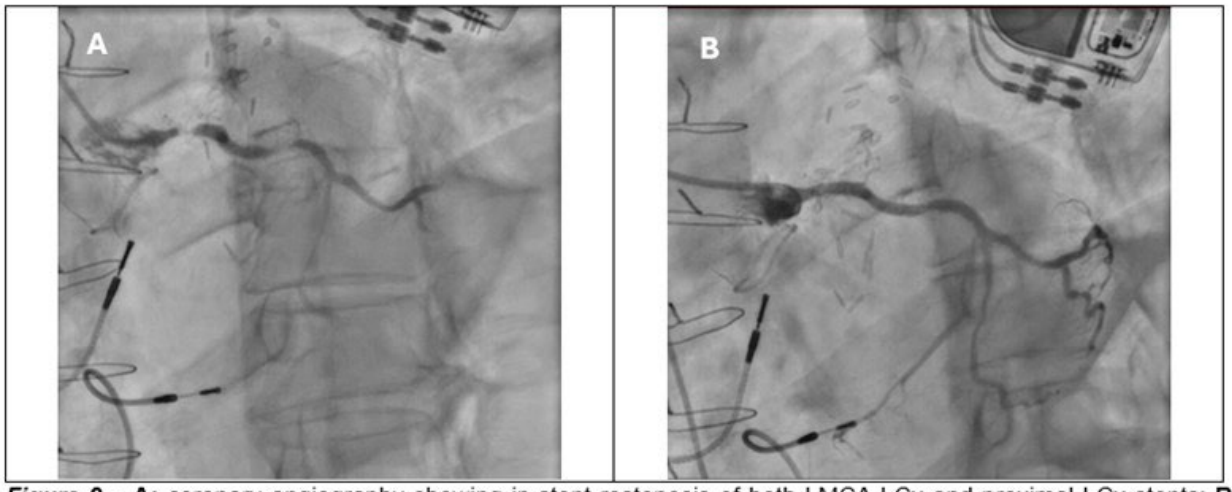

Figure 2 – **A:** coronary angiography showing in-stent restenosis of both LMCA-LCx and proximal LCx stents; **B:** final angiographic results following percutaneous coronary intervention (PCI).

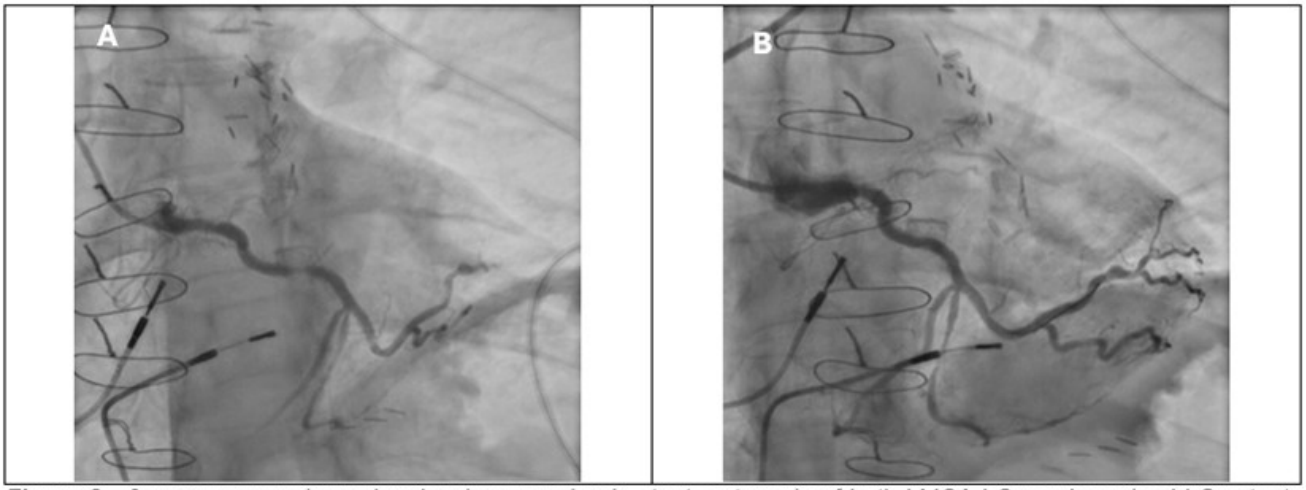

Figure 3 - **A:** coronary angiography showing recurring in-stent restenosis of both LMCA-LCx and proximal LCx stents following PRRT treatment; **B:** final angiographic results following drug-eluting balloon angioplasty.

When jaundice reveals a cardiac complication: severe haemolytic anaemia secondary to mitral paravalvular leak

Viviana Ferreira[1]; Inês Barroso Almeida[1]; Margarida Câmara Farinha[1]; Inês Coutinho Dos Santos[1]; Maria Inês Barradas[1]; Ricardo Carvalheiro[2]; António Fontes[1]; Carina Machado[1]; Luís Oliveira[1]; António Fiarresga[2]; Miguel Pacheco[1]

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Introduction

Paravalvular leak (PVL) is a recognized complication following prosthetic valve implantation and may remain clinically silent for years. Significant PVL can lead to severe haemolytic anaemia and heart failure, posing important diagnostic and therapeutic challenges. We report a case of severe haemolysis caused by mitral prosthetic valve dysfunction, presenting predominantly with jaundice.

Case Description

A 79-year-old woman with a history of mechanical mitral valve replacement was admitted to the Emergency Department with progressive jaundice. She had a recent hospitalization in the Haematology Department for investigation of direct antiglobulin test-negative haemolytic anaemia. Previous work-up, including abdominal computed tomography, direct and indirect Coombs tests and evaluation for alternative causes of haemolysis, was unremarkable. Electrocardiogram showed sinus rhythm without ischemic abnormalities. Laboratory investigation revealed severe macrocytic anaemia, elevated lactate dehydrogenase (3148U/L), indirect hyperbilirubinemia (1.87 mg/dL), reticulocytosis (14.6%) and haptoglobin decreased (<0.03 g/dL), consistent with haemolytic anaemia.

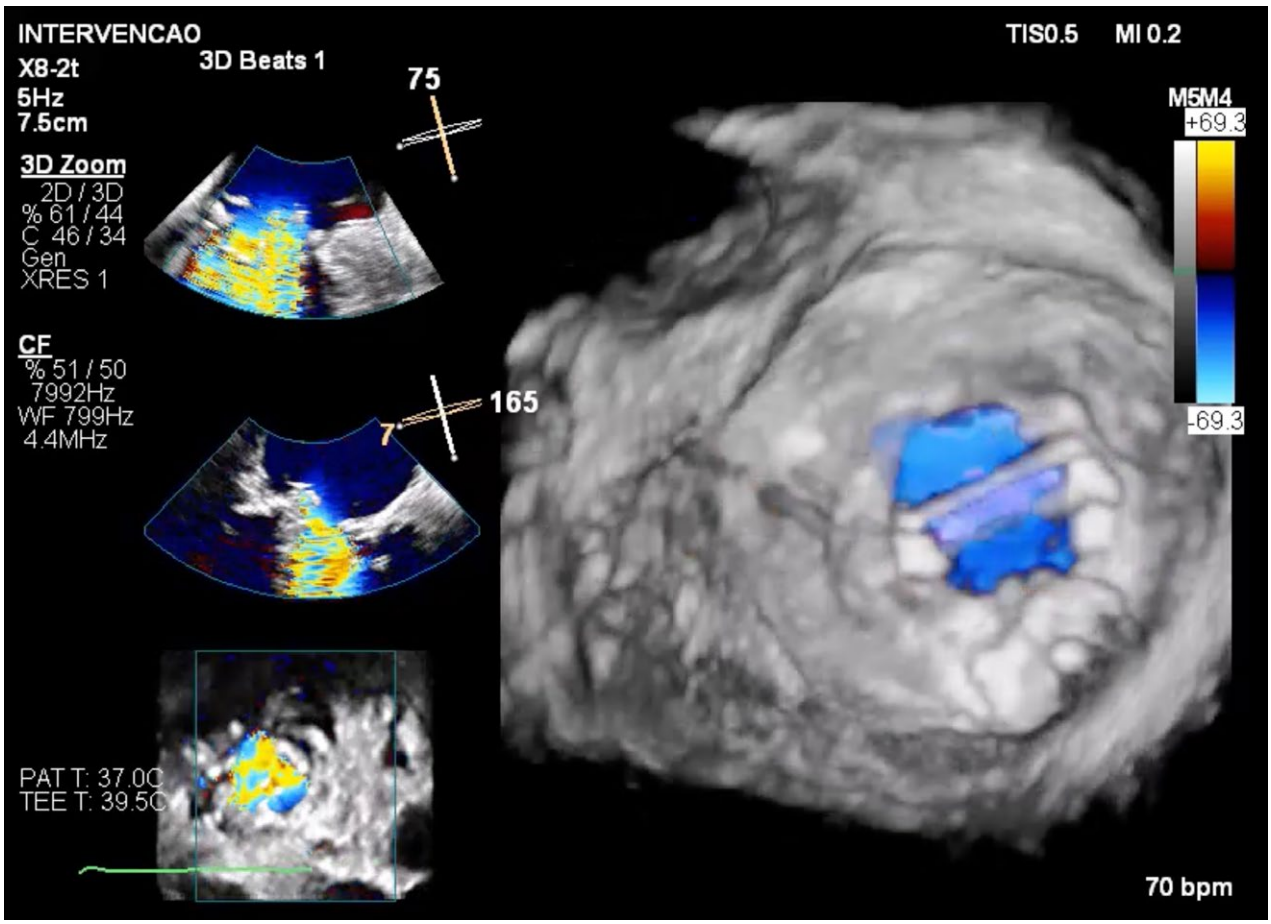
Given the history of prosthetic valve replacement, mechanical haemolysis was suspected. Transthoracic echocardiography revealed increased transprosthetic gradients, raising suspicion of prosthetic valve dysfunction. Transoesophageal echocardiography demonstrated preserved prosthetic disc mobility and a moderate paravalvular mitral regurgitation with high velocity flow caused by posteromedial annular dehiscence/pseudoaneurysm.

After Heart Team discussion, the patient was referred to a tertiary centre for percutaneous closure of peri-prosthetic leak. The postoperative period was complicated by cardiac tamponade requiring urgent surgical intervention.

Following clinical stabilization, we observed progressive improvement of haemolytic parameters and jaundice. The patient is currently under regular outpatient cardiology follow-up, with favourable clinical evolution.

Discussion

Although frequently asymptomatic, significant mitral PVL may present with severe haemolytic anaemia and non-specific systemic manifestations such as jaundice, delaying diagnosis. In patients with prosthetic valves presenting with haemolytic anaemia, prosthesis-related mechanical haemolysis should always be considered. Multimodality cardiac imaging, particularly transoesophageal echocardiography, plays a central role in identifying prosthetic dysfunction, assessing severity and guiding therapeutic strategy. This case highlights the importance of early recognition and multidisciplinary management in complex valvular complications.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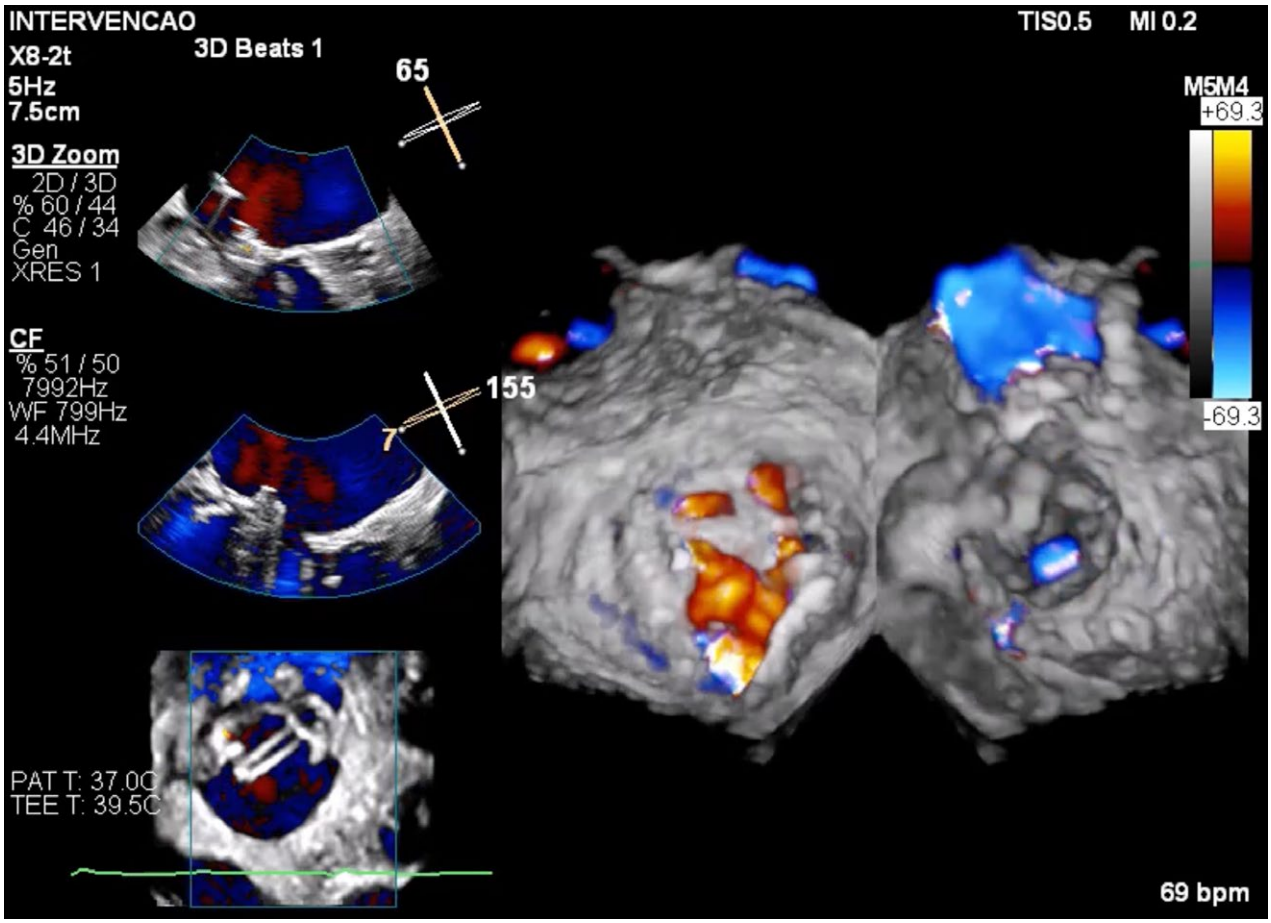
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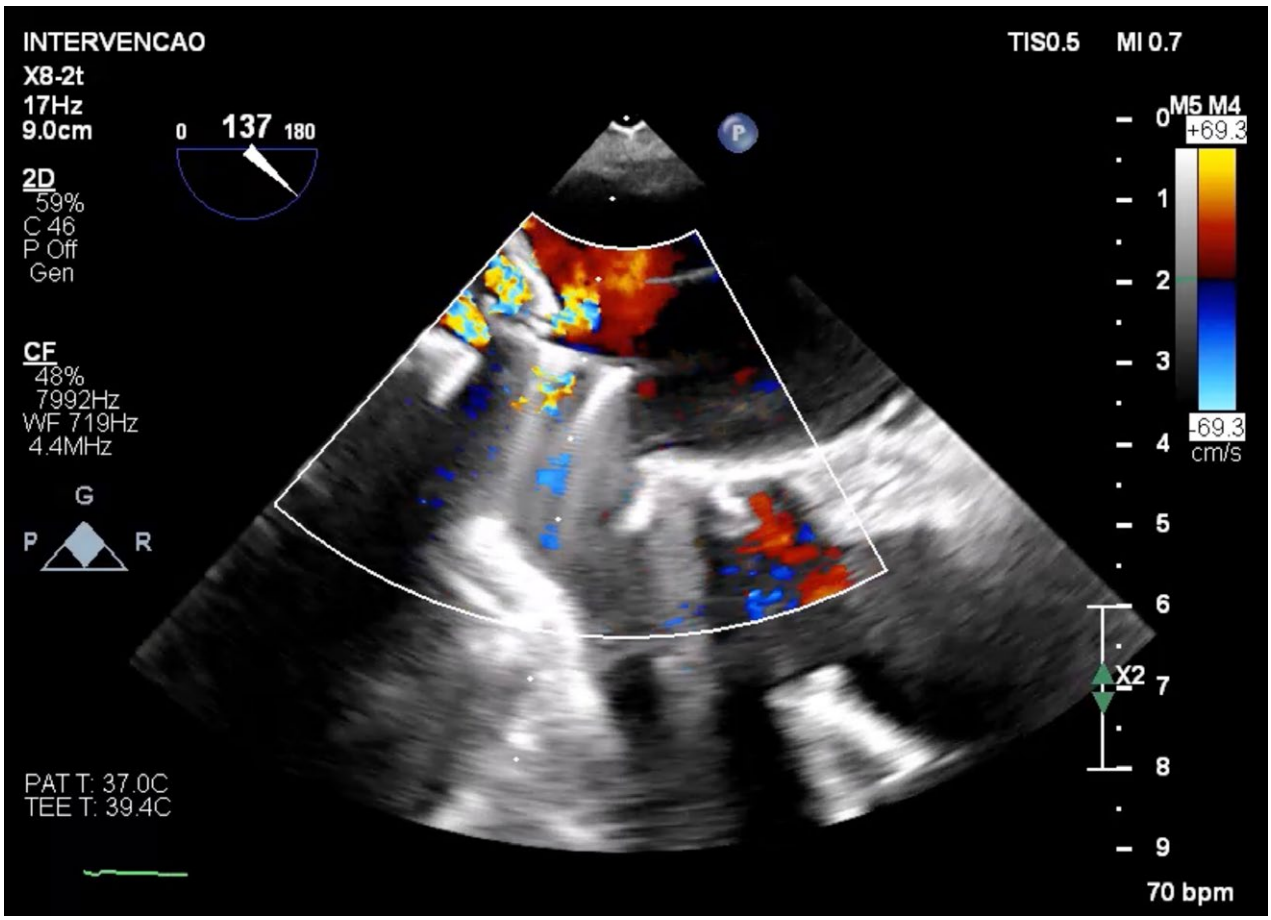
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Driven by Infection, Defined by Innovation: A New Path in Cardiac Device Therapy

Andreia Lopes Sousa; Bárbara Rocha; João Faria; Carla Oliveira Ferreira; Filipe Vilela; Mónica Dias; Sofia Fernandes; Carla Marques Pires; Catarina Vieira; Rui Files Flores

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

Endocarditis involving cardiac implantable electronic devices are associated with high morbidity and complex management, particularly when metastatic infectious foci coexist with the need for device extraction and reimplantation.

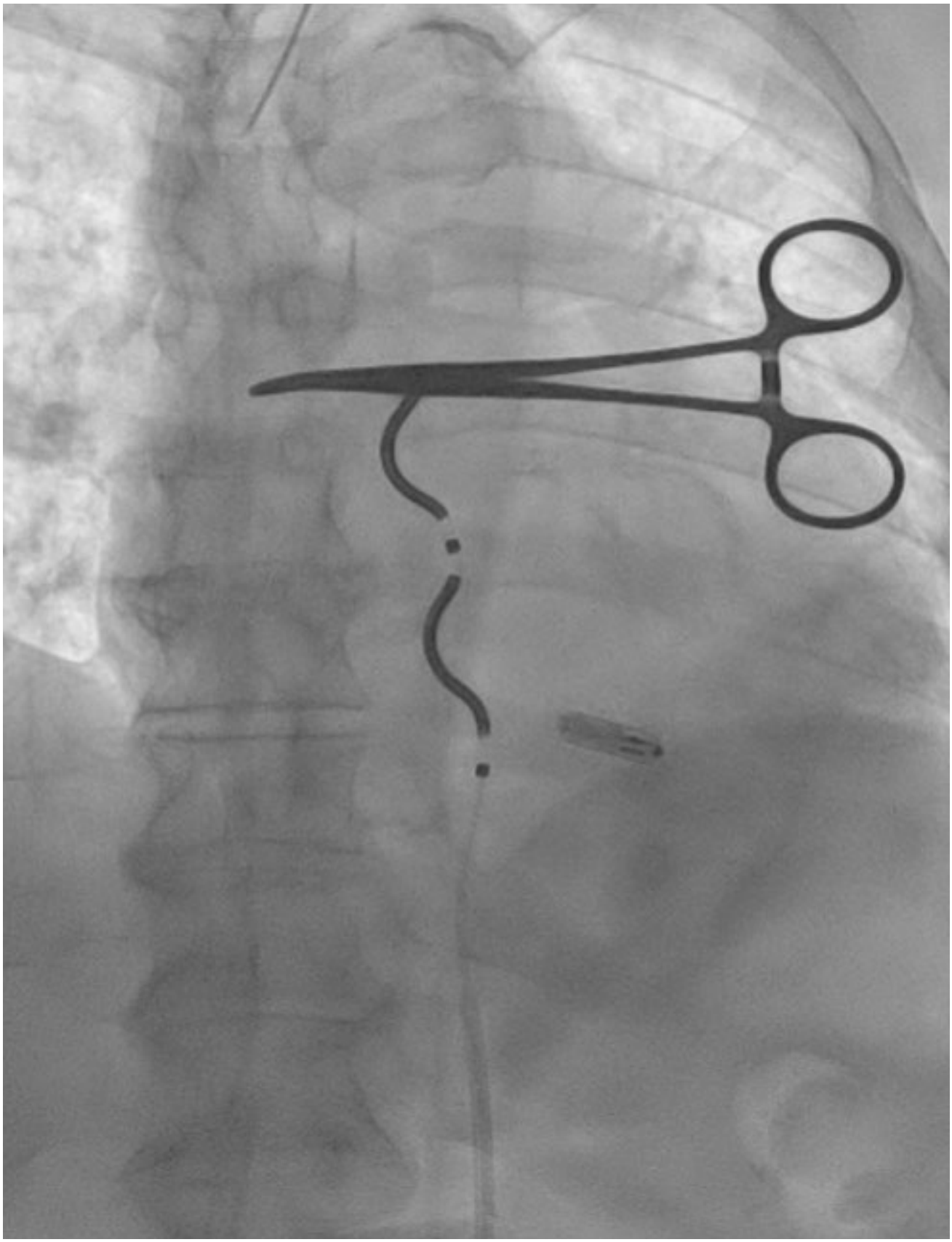
Case presentation

A 76-year-old man presented with 3 days of cervical pain, fever, and acute lower limb motor deficit. His history included hypertension, type 2 diabetes mellitus, and a permanent pacemaker implanted 15 years earlier for complete atrioventricular block. Abdominal CT showed emphysematous pyelonephritis. Neuroimaging revealed multiple abscesses in the spinal canal and cervical musculature causing spinal cord compression, requiring urgent neurosurgery. Cultures from pus and urine grew Escherichia coli. During hospitalization, ankle pain led to the diagnosis of osteomyelitis with the need for debridement surgery, supporting disseminated infection. Transesophageal echocardiography identified a 13 mm pacemaker lead vegetation, establishing device-related infective endocarditis. After targeted antibiotic therapy, the pacing system was extracted and replaced with a leadless pacemaker.

The course was complicated by new and recurrent Klebsiella pneumoniae bacteraemia; PET-CT was used to exclude leadless pacemaker infection. After more than 6 months of hospitalization, the patient suffered a cardiac arrest with a shockable rhythm. Post-resuscitation ECG showed anterolateral ST elevation. Coronary angiography revealed extensive coronary disease with chronic proximal circumflex occlusion with a moderate systolic dysfunction on echocardiography. Cardiac MRI confirmed a lateral wall infarction scar consistent with an arrhythmogenic substrate. As no other reversible cause was identified, a dysrhythmic event was assumed. Due to inadequate sensing for a standard subcutaneous device and the infection risk of a transvenous system, a retrosternal subcutaneous cardioverter-defibrillator (Aurora system) was implanted for secondary prevention. The patient was discharged in stable condition.

Discussion

This case underscores the complexity of cardiac device selection and the potential for complications, while emphasizing the importance of timely device extraction and source control in the management of infection. It also highlights the challenges associated with reimplantation decisions and arrhythmic risk management, requiring a careful balance between infection prevention and ongoing protection from life-threatening arrhythmias.



When the Gene Is Unclear: Familial Bicuspid Aortic Valve, Aortopathy and a Possible New Variant

Andreia Lopes Sousa; João Faria; Bárbara Rocha; Carla Oliveira Ferreira; Filipe Vilela; Mónica Dias; Sofia Fernandes; Carla Marques Pires; Rui Files Flores

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

Bicuspid aortic valve is a common congenital cardiac anomaly frequently associated with aortopathy and, in some cases, a familial pattern of inheritance. Identification of pathogenic or likely pathogenic variants may help clarify disease mechanisms, guide surveillance, and support family screening. We report a case of asymptomatic bicuspid aortic valve with significant thoracic aortic dilation and two variants of uncertain significance in genes associated with familial aortopathy, raising the possibility of a novel genetic contribution.

Case presentation

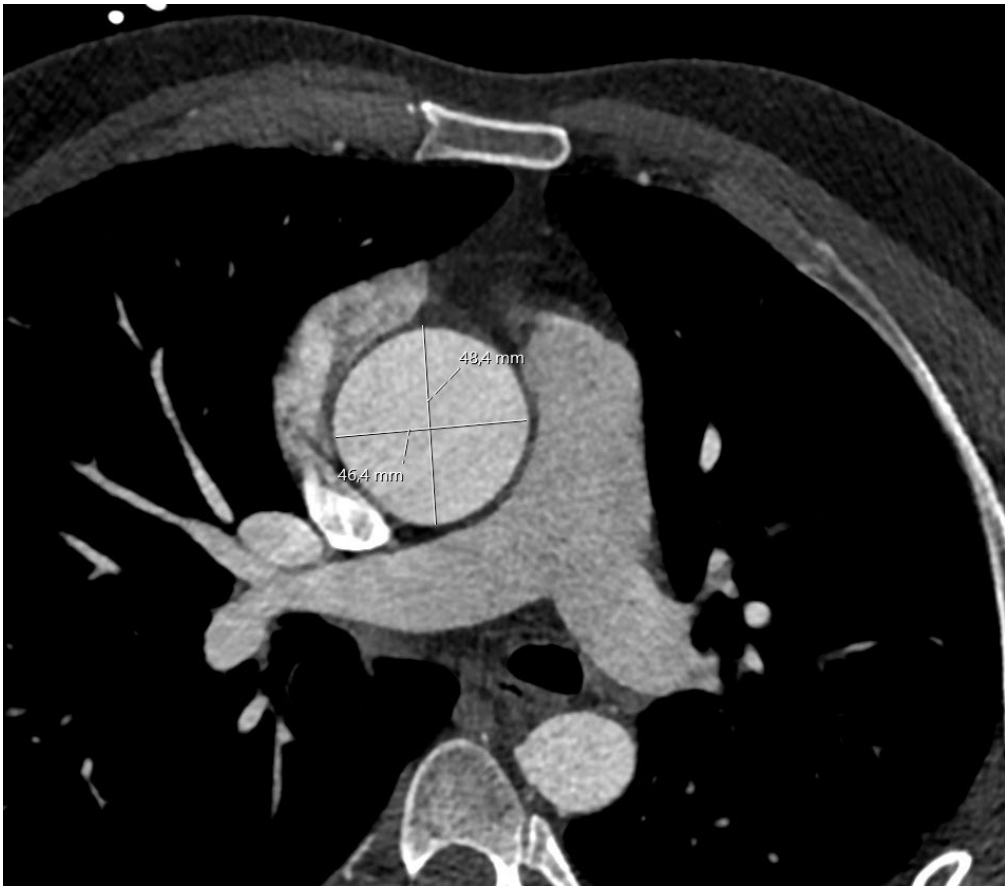
A 43-year-old man was referred for cardiology evaluation and screening for aortic disease in the context of a relevant family history, including a cousin who had undergone surgery for an ascending aortic aneurysm and bicuspid aortic valve. The patient was asymptomatic and had no previous medical history. Physical examination revealed a decrescendo diastolic murmur at the left sternal border.

Transthoracic echocardiography and CT angiography demonstrated a bicuspid aortic valve of Sievers type 1 R-L, with mild cusp thickening and moderate aortic regurgitation, associated with marked dilation of the aortic root and ascending aorta (48 mm. Coronary calcium score and aortic valve calcium score were both 0 Agatston units. The remaining aorta showed only mild distal dilation.

Genetic testing for familial aortopathy identified two variants of uncertain significance: FBN2 c.566G>A p.(Arg189His) and NOTCH1 c.5572A>C p.(Met1858Leu), both in heterozygosity. Further family evaluation and cascade screening was initiated, including proceeding to genotyping our patient's cousin.

Discussion

This case highlights the importance of systematic family screening in patients with bicuspid aortic valve and thoracic aortopathy, particularly when a suggestive family history is present. The identification of previously uncharacterized NOTCH1 and FBN2 variants, together with aortic dilation and valve dysfunction, raises the possibility of a novel genetic contribution to the phenotype. Segregation analysis and extended familial evaluation will be essential to clarify variant significance and support future reclassification.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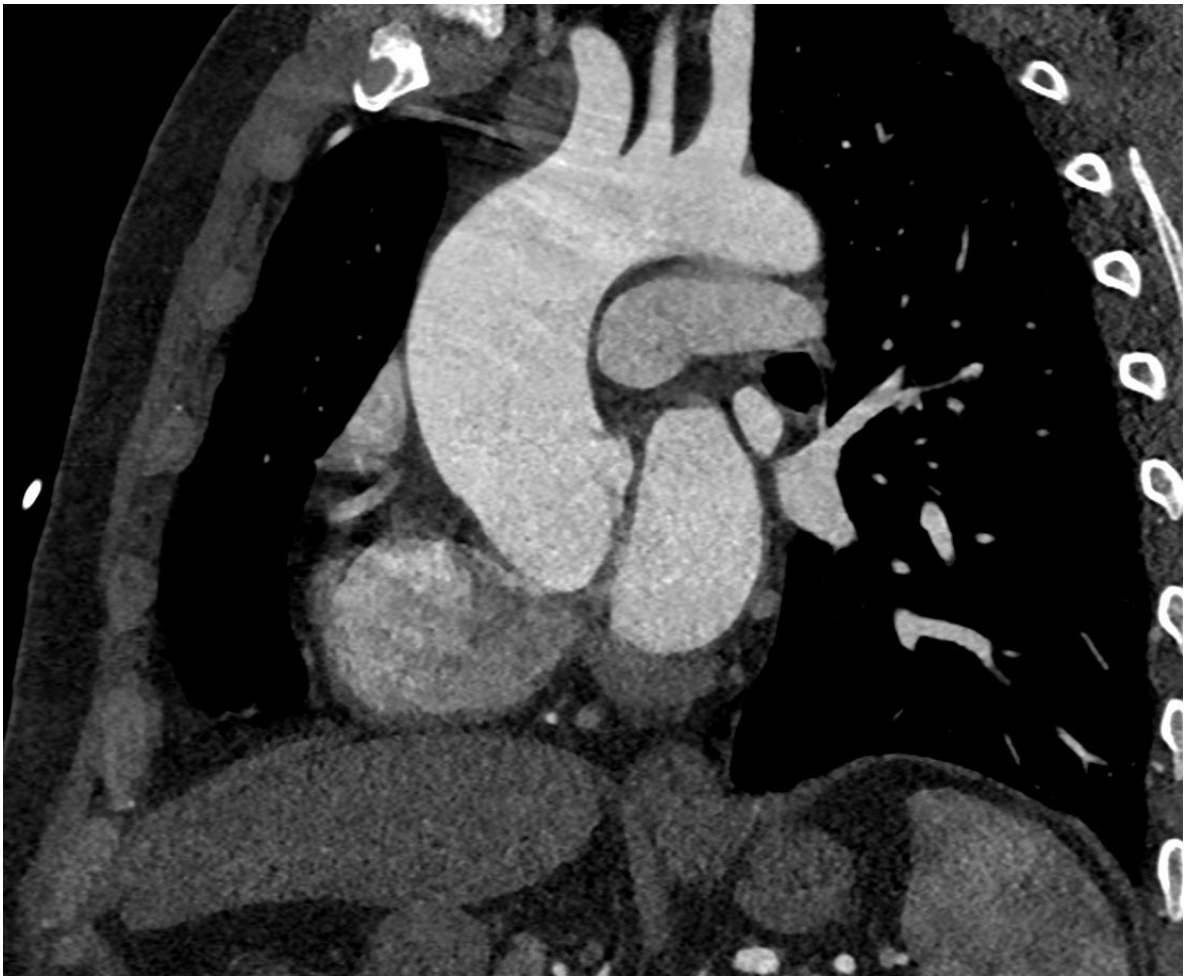


From Negative to Definitive: How Endomyocardial Biopsy Unlocked Systemic AL Amyloidosis

Andreia Lopes Sousa; Carla Oliveira Ferreira; Bárbara Rocha; João Faria; Filipe Vilela; Sofia Fernandes; Mónica Dias; Rui Files Flores

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

Systemic AL amyloidosis is a challenging diagnosis because clinical manifestations are often nonspecific and organ involvement can be patchy or absent on initial biopsies. In such settings, endomyocardial biopsy can play a decisive role in establishing the diagnosis when other organ biopsies are non-diagnostic and suspicion for AL amyloidosis remains high.

Case presentation

A 67-year-old woman with a history of indolent light-chain multiple myeloma and chronic kidney disease, attributed to hypertensive nephropathy, was admitted for investigation of hepatic cholestasis. Obstructive, infectious, and toxic causes were excluded. Renal and hepatic biopsies had shown no apparent amyloid deposits. Although she was paucisymptomatic from a cardiac standpoint, transthoracic echocardiography revealed findings suggestive of infiltrative cardiomyopathy, with marked biventricular hypertrophy and preserved global biventricular systolic function. In the setting of plasma cell dyscrasia, renal and hepatic injury, and a suggestive echocardiographic phenotype, AL amyloidosis was strongly suspected. Cardiac DPD scintigraphy was negative, but diagnostic suspicion remained high. Endomyocardial biopsy was then performed and revealed deposition of amorphous material with Congo red birefringence, confirming AL cardiac involvement and establishing the diagnosis of systemic AL amyloidosis as the unifying explanation for the cardiac, renal, and hepatic involvement. After one year of chemotherapy, the patient remains clinically stable.

Discussion

This case highlights the critical role of endomyocardial biopsy in diagnosing systemic AL amyloidosis when other organ biopsies are non-diagnostic and noninvasive testing is inconclusive. In patients with plasma cell dyscrasia and multiorgan injury, a negative scintigraphy and minimal or absent cardiac symptoms should not deter further investigation when suspicion for AL amyloidosis persists, as endomyocardial biopsy may be essential to reveal the underlying systemic diagnosis and guide disease-directed therapy. Advances in hematologic treatments seem to be altering the expected lifespan of these patients.

Mysteries around an overwhelmed right ventricle: Severe Primary Pulmonary Regurgitation Mimicking Pulmonary Hypertension

Andreia Lopes Sousa; Mónica Dias; Bárbara Rocha; João Faria; Filipe Vilela; Carla Oliveira Ferreira; Sofia Fernandes; Carla Marques Pires; Catarina Vieira; Rui Files Flores

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

Pulmonary hypertension is a common cause of right ventricular overload and dilatation, particularly in women with autoimmune or connective tissue disease, in whom it is a frequent diagnostic consideration. However, right heart strain may also result from primary valvular disease or late sequelae of congenital heart interventions.

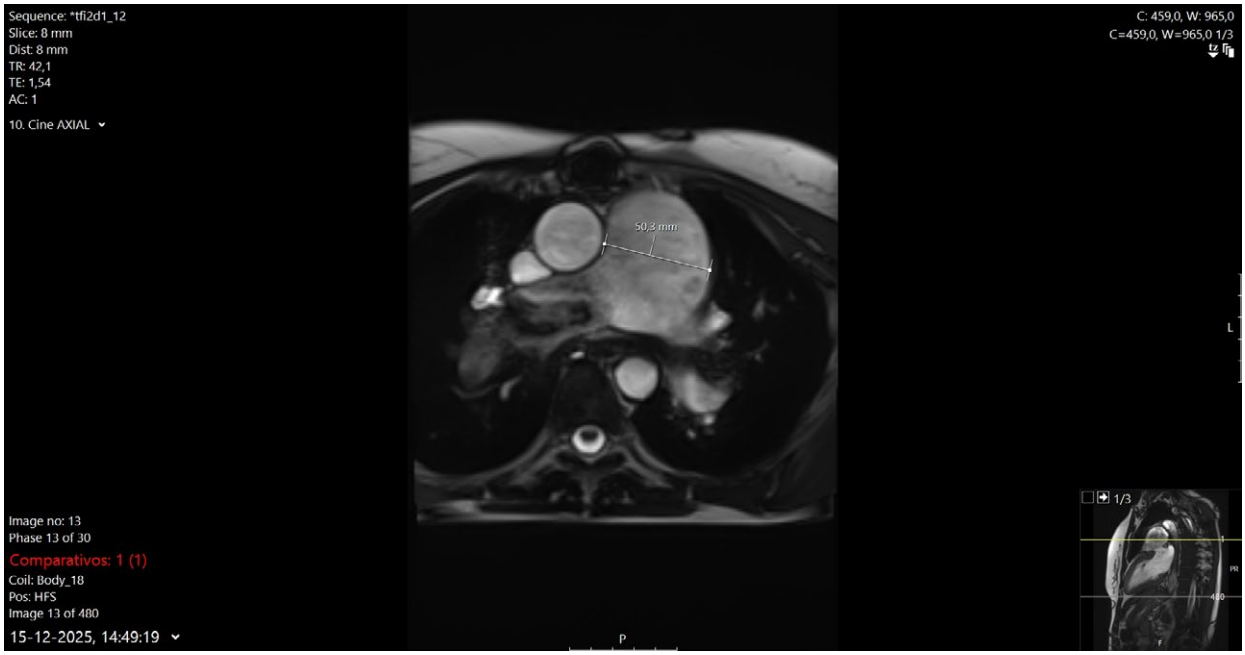
Case presentation

A 49-year-old woman was referred for cardiology assessment because of suspected pulmonary hypertension during investigation for a connective tissue disease, with suspected lupus. Her medical history included surgical closure of an interatrial communication in infancy. Transthoracic echocardiography showed moderate right ventricular dilatation, dilatation of the pulmonary trunk (37 mm), tricuspid annular dilatation (43 mm) with moderate tricuspid regurgitation, and an estimated systolic pulmonary artery pressure of 39 mmHg. Cardiac magnetic resonance confirmed severe right ventricular dilatation, pulmonary trunk dilatation up to 48 mm, and interventricular septal flattening consistent with right-sided volume overload. Transoesophageal echocardiography showed no residual interatrial communication but demonstrated severe pulmonary regurgitation. Right heart catheterization excluded with pulmonary hypertension.

Further gathering of medical registries revealed prior balloon dilatation for congenital pulmonary valve stenosis at 5 years of age. The overall findings were consistent with chronic severe pulmonary regurgitation as the cause of right ventricular overload and dilatation, a recognized late consequence after pulmonary valvuloplasty in some patients.

Discussion

This case illustrates that right ventricular overload and dilatation are not synonymous with pulmonary hypertension, even in a patient profile in which pulmonary hypertension is strongly suspected. In patients with previous congenital heart interventions or known pulmonary valve disease, severe pulmonary regurgitation should be considered in the differential diagnosis, and right heart catheterization and advanced imaging techniques remain the gold standard to exclude pulmonary hypertension and avoid misdiagnosis.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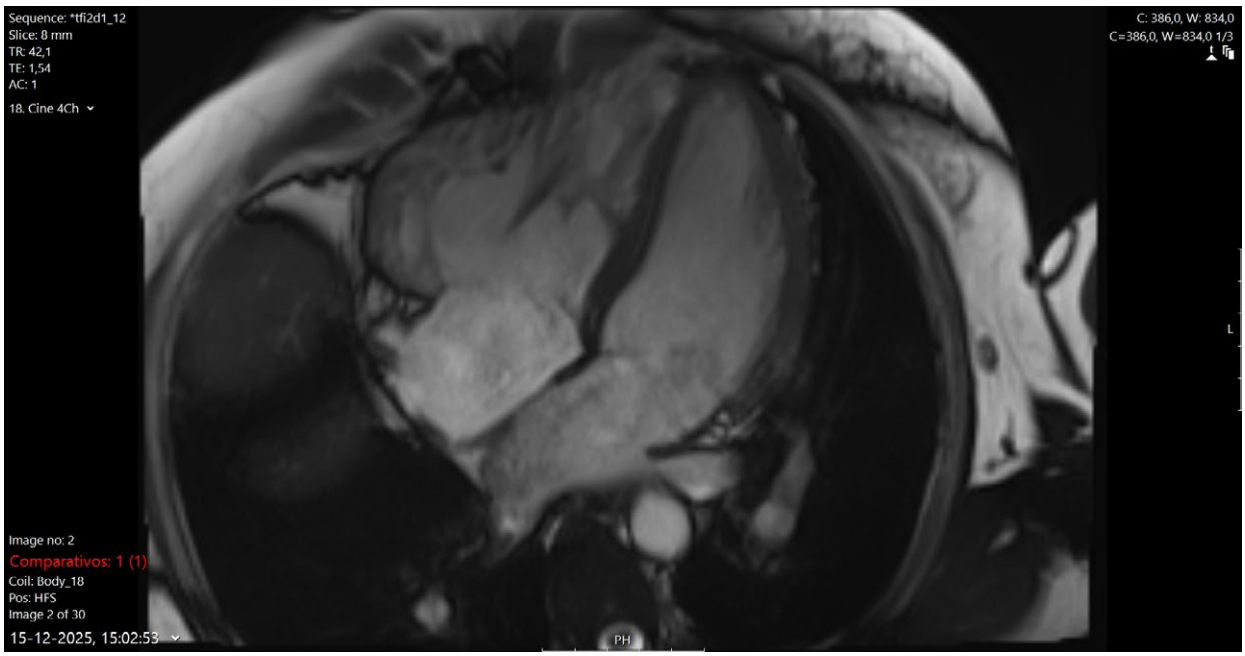
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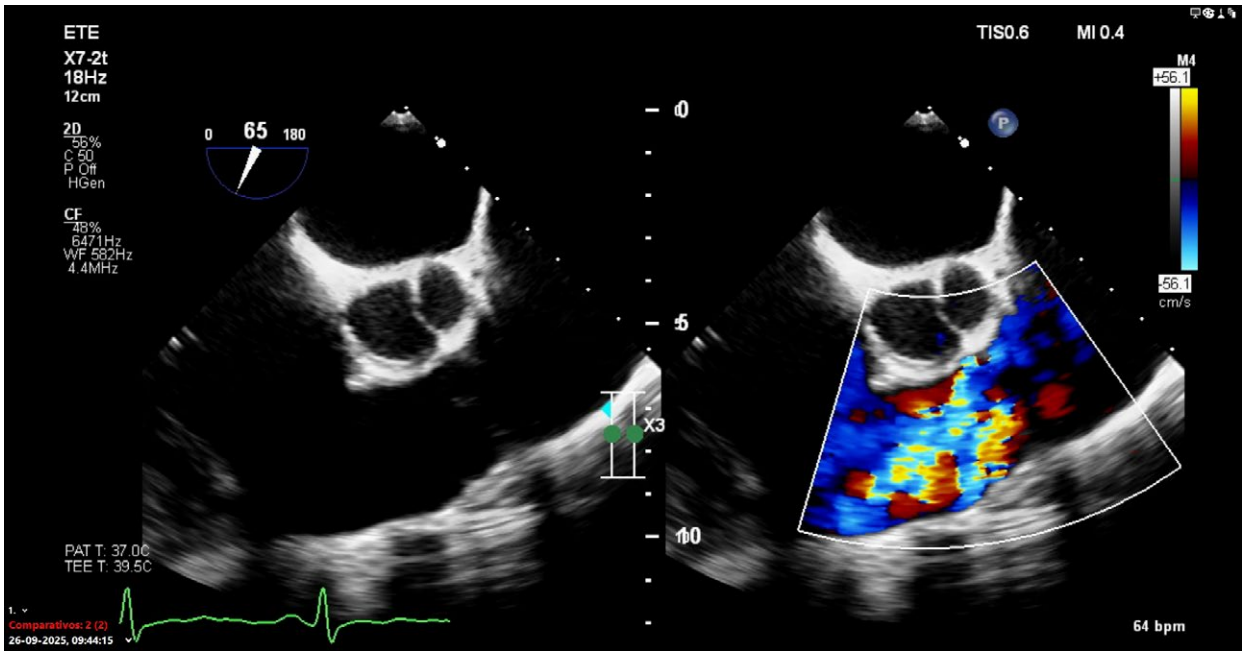
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Normotensive Shock in Acute Pulmonary Embolism: The Importance of Early Recognition of Occult Hypoperfusion

Bárbara Antunes Rocha; João Faria; Andreia Sousa; Carla Ferreira; Filipe Vilela; Mónica Dias; Sofia Fernandes; Carlos Galvão Braga; Rui Files Flores

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

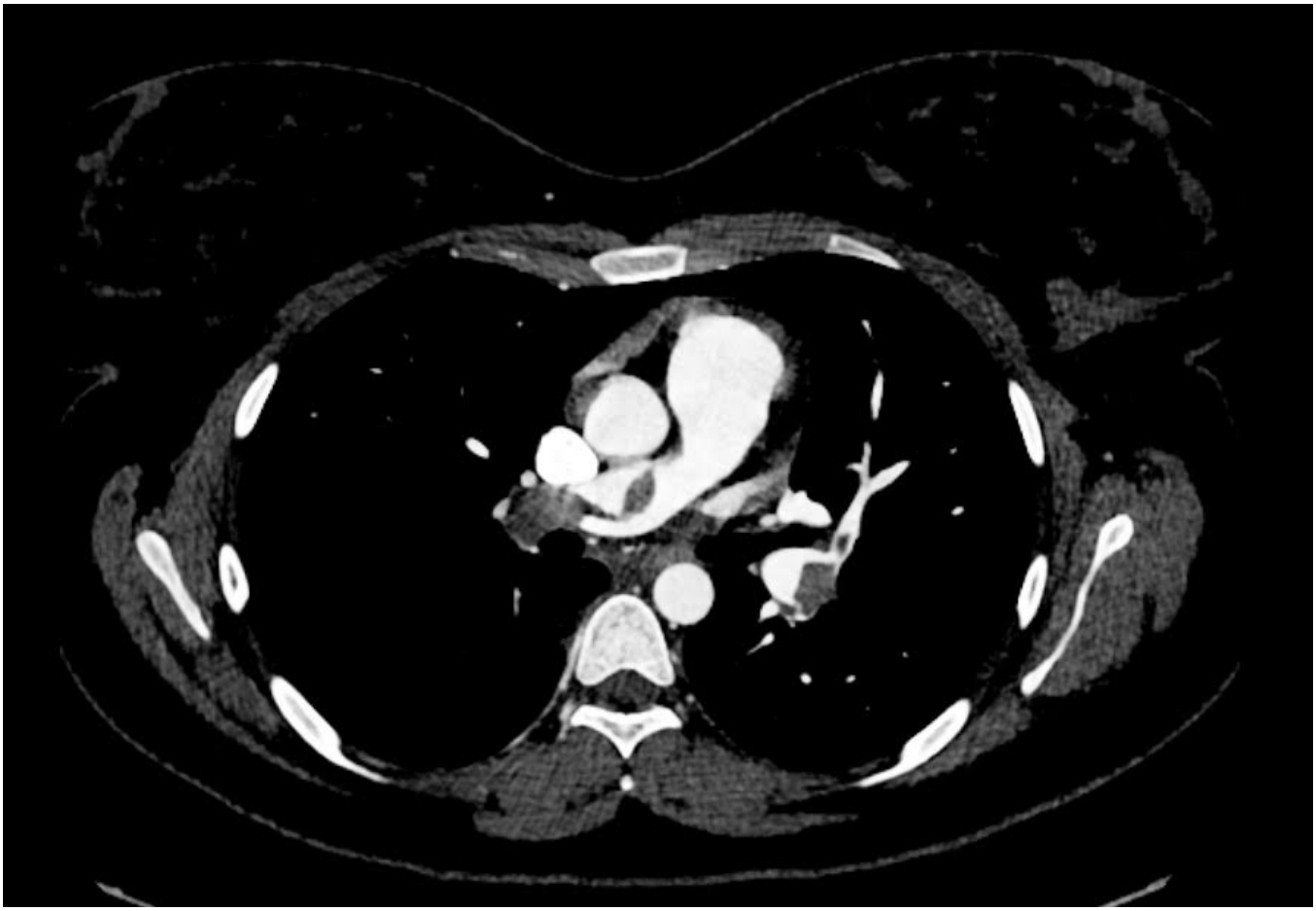
Pulmonary embolism (PE) is a life-threatening cardiovascular emergency with a variable clinical presentation, ranging from mild dyspnea to circulatory collapse. Although more commonly associated with older patients and multiple comorbidities, severe PE may also occur in young individuals when acquired prothrombotic factors coexist. Combined oral contraceptive use and obesity are recognised contributors to thromboembolic risk. Early recognition of haemodynamic compromise and right ventricular (RV) dysfunction is essential, as delayed escalation of reperfusion therapy may adversely affect outcomes. Clinical progression, clot burden, and imaging markers of RV overload are key determinants in identifying patients requiring escalation beyond anticoagulation, namely in the onset of normotensive shock.

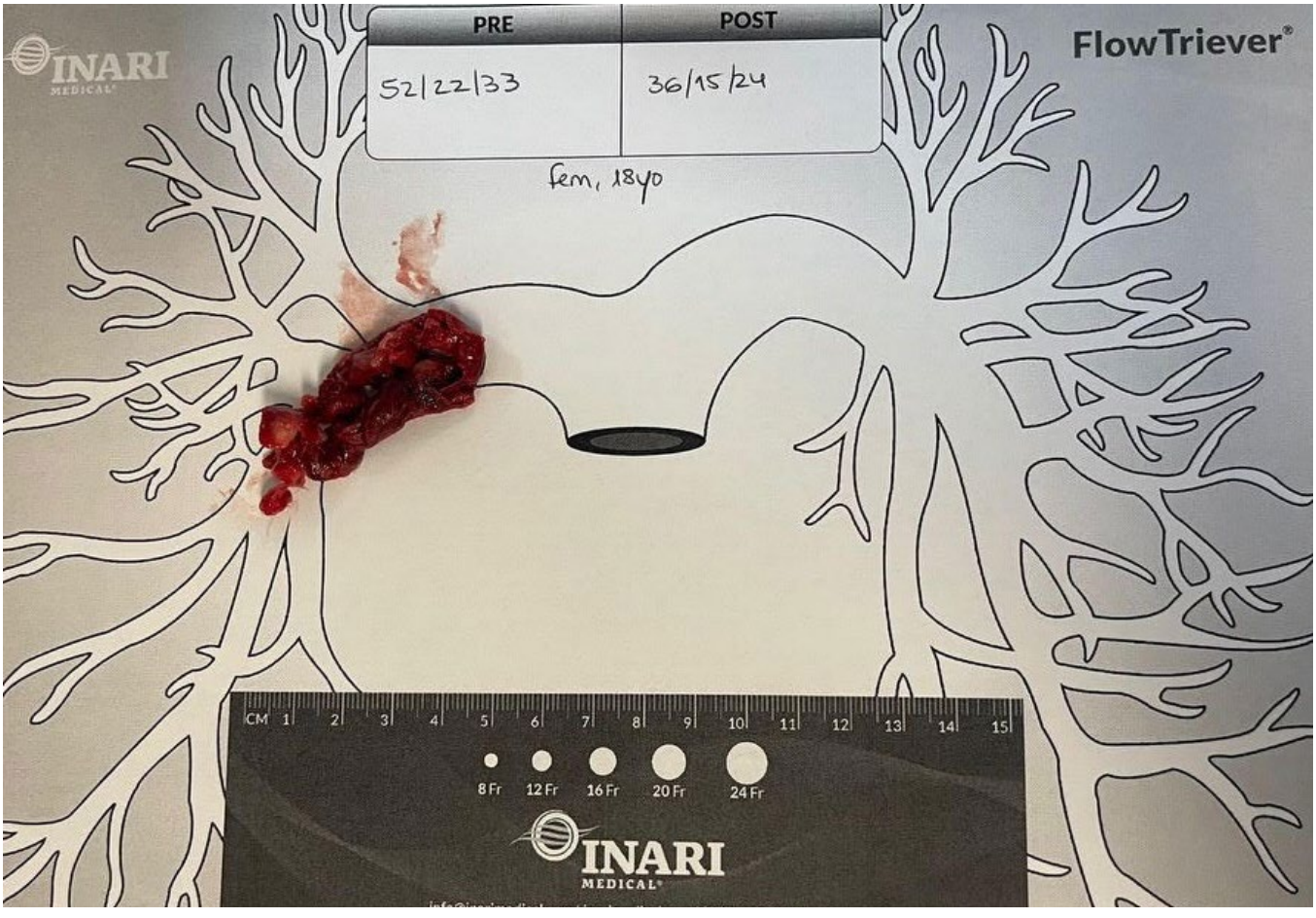
Case report

An 18-year-old woman with obesity and anxiety disorder, under combined oral contraceptive, presented to the emergency department after syncope. On admission, she was normotensive, tachycardic, hypoxaemic, and exhibited signs of peripheral hypoperfusion with elevated lactate (sPESI ≥1; PESI 108, Class IV). ECG demonstrated sinus tachycardia with the classic S1Q3T3 pattern. Cardiac biomarkers were elevated (NT-proBNP 3404 pg/mL; Troponin I 0.813 ng/mL). Transthoracic echocardiography revealed a dilated and dysfunctional right ventricle, interventricular septal flattening, shortened pulmonary artery acceleration time (50 ms), and estimated sPAP of 55 mmHg. CTPA demonstrated extensive bilateral PE involving lobar and segmental branches of the right lung and left lower lobe, with a saddle thrombus at the right main pulmonary artery and high clot burden (CT obstruction index >40%). The patient was admitted to the ICU where, despite anticoagulation, she developed worsening respiratory failure with increasing oxygen requirements. Repeat CTPA demonstrated thrombus propagation into bilateral segmental and subsegmental branches. Following multidisciplinary PERT discussion, she underwent urgent percutaneous thrombectomy (Flowtriever 24 Fr, right femoral venous access), with retrieval of a large clot burden and immediate haemodynamic normalisation.

Conclusion

This case highlights severe PE in a young woman with identifiable yet underestimated thrombotic risk factors. Despite early recognition, normotensive shock was likely overlooked, delaying timely percutaneous intervention. This underscores the importance of PERT activation and consideration of advanced reperfusion strategies in selected patients. The case further illustrates the nature of severe PE and the limitations of isolated risk stratification tools in predicting deterioration. Integration of clinical severity, RV dysfunction, biomarker elevation, clot burden, and clinical evolution may facilitate earlier recognition and more timely intervention.

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A Reversible Cause, an Irreversible Block: Diagnostic Challenges in Complete Atrioventricular Block

Bárbara Antunes Rocha; Andreia Sousa; João Faria; Carla Ferreira; Filipe Vilela; Mónica Dias; Sofia Fernandes; Catarina Vieira; Carlos Galvão Braga

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

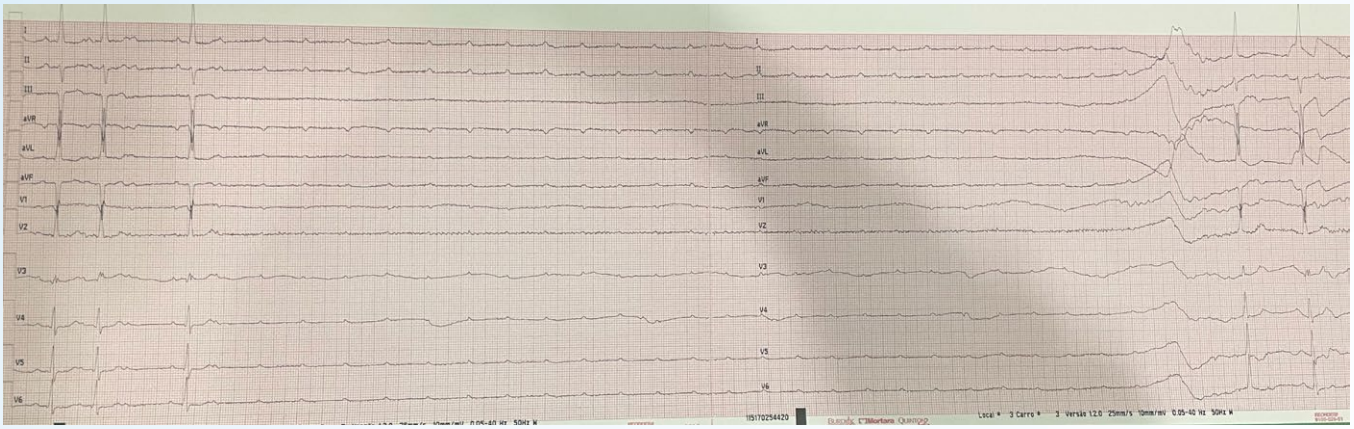
Complete atrioventricular block (CAVB) is characterised by absent atrioventricular conduction, resulting in severe bradycardia, syncope, and sudden cardiac death. In young patients, CAVB is uncommon and requires extensive investigation, as reversible causes including electrolyte disturbances, drug toxicity, and endocrine disorders such as hypothyroidism must be excluded before permanent pacing is considered. Although hypothyroidism is a recognised reversible cause of conduction disease, persistence of CAVB despite hormonal correction should prompt investigation for structural or idiopathic disease. CMR enables tissue characterisation and identification of otherwise occult abnormalities.

Case Report

A 38-year-old woman with obesity and previous total thyroidectomy for multinodular goitre, without current medication or relevant family history, presented with recurrent syncope. On admission, she was normotensive with a heart rate of 30 bpm. ECG monitoring documented CAVB with a ventricular pause exceeding 20 seconds and convulsive syncope. Isoprenaline achieved suboptimal chronotropic response, requiring urgent temporary transvenous pacing. Laboratory tests showed normal electrolytes and hypothyroidism (TSH 74 mIU/L; free T4 1.25 pmol/L). The patient remained pacemaker-dependent throughout. A long-term temporary pacemaker was implanted and levothyroxine initiated. CMR showed a non-dilated left ventricle with mild basal septal hypertrophy, mildly reduced systolic function in the context of pacing-induced dyssynchrony, prominent trabeculation of the anterolateral and apical segments, myocardial crypts in the basal anterolateral and inferolateral walls, and non-ischaemic mid-wall fibrosis in the basal anterolateral and inferolateral segments with an additional focus at the inferior interventricular junction. Following thyroid function normalisation without recovery of conduction, left bundle branch pacing was selected to minimise long-term dyssynchrony. Genetic testing is pending.

Conclusion

This case highlights the complexity of CAVB in a young patient. Despite hypothyroidism as a potentially reversible cause, persistence of conduction block after hormonal correction suggested an underlying cardiomyopathic substrate, supported by the CMR findings and warranting genetic investigation. The multidisciplinary stepwise approach encompassing aetiological investigation, temporary pacing, CMR characterisation, and optimisation of pacing modality was central to management. This case underscores the importance of comprehensive investigation and advanced imaging in young patients with CAVB, even when an apparent reversible cause is identified.



The Storm Behind the Shock: Acute Biventricular Failure in Undiagnosed Graves' Disease

Alexandru Babiciu; Filipa Saraiva; Hugo Costa; Raquel Fernandes; Dina Bento; Lucas Hamann; Joana Guerreiro Pereira; Sofia Andraz; Joana Massa Pereira; Jorge Mimoso

UNIDADE LOCAL DE SAÚDE DO ALGARVE

Introduction

Thyroid storm is a rare endocrine emergency associated with severe multisystem dysfunction and mortality rates of 10-20%. Although cardiovascular involvement is frequent, progression to severe biventricular dysfunction with cardiogenic shock is uncommon and may delay diagnosis.

We report a case of thyrotoxic crisis secondary to Graves' disease presenting as refractory acute heart failure and cardiogenic shock.

Case Presentation

A 48-year-old male with no previous medical history presented to the emergency department with 24 hours of dyspnea, palpitations, and chest discomfort. On admission, he was delirious, diaphoretic, flushed, tachypneic, hypoxemic, and hypotensive (74/61 mmHg), with pulmonary congestion and peripheral edema ECG showed atrial fibrillation with rapid ventricular response at 200 bpm.

Acute heart failure with cardiogenic shock secondary to tachycardiomyopathy was initially suspected.

Hemodynamic instability, led to urgent electrical cardioversion (100 J), followed by invasive mechanical ventilation and ICU admission.

Transthoracic echocardiography demonstrated severe biventricular dysfunction with left ventricular dilation, diffuse hypokinesia, LVEF 15-20%, moderate mitral and tricuspid regurgitation and a dilated inferior vena cava.

Laboratory evaluation showed elevated NT-proBNP (6611 pg/mL), mildly increased hs-cTnI (41.7 pg/mL), and marked hepatic cytolysis (AST 1837 U/L; ALT 1216 U/L). Viral serologies, blood cultures, and autoimmune studies were negative.

In the ICU, the patient developed persistent fever, profuse sweating, warm extremities, and refractory cardiogenic shock despite cardioversion and escalating vasoactive support with dobutamine and norepinephrine, raising suspicion of a hypermetabolic state.

Progressive hemodynamic deterioration prompted transfer to a tertiary center for mechanical circulatory support.

Thyroid function tests later confirmed severe thyrotoxicosis with suppressed TSH (<0.005 mUI/L), elevated FT4 and FT3, and positive TRABs, consistent with Graves' disease.

Treatment with methimazole, hydrocortisone, and propranolol was initiated alongside standard heart failure therapy, ventilatory support, and vasoactive therapy.

Progressive clinical improvement occurred, and after 7 days the patient was extubated, weaned from inotropes, and transferred back to the referring center.

Follow-up echocardiography showed partial recovery of biventricular function with LVEF of 31%.

Discussion

Thyroid storm is an uncommon but critical cause of acute biventricular failure and refractory cardiogenic shock, requiring high clinical suspicion for timely diagnosis.

Early multidisciplinary management with antithyroid therapy, corticosteroids, beta-blockade, and advanced circulatory support is essential for hemodynamic stabilization and recovery of ventricular function.

When Lupus Strikes the Heart: Acute Heart Failure and Complete Atrioventricular Block as the First Manifestation of SLE

Alexandru Babiciu; Filipa Saraiva; Hugo Costa; Raquel Fernandes; Dina Bento; Joana Guerreiro Pereira; Lucas Hamann; Sofia Andraz; Joana Massa Pereira; Jorge Mimoso

UNIDADE LOCAL DE SAÚDE DO ALGARVE

Introduction

Lupus myocarditis is a rare but severe manifestation of systemic lupus erythematosus (SLE), particularly when cardiac involvement precedes systemic disease recognition. Clinical presentation ranges from asymptomatic ventricular dysfunction to acute heart failure (AHF) and malignant arrhythmias.

Case Report

A 36-year-old woman with antiphospholipid syndrome on apixaban was admitted for distal ischemia of the left lower limb. During hospitalization, she developed acute dyspnea, chest discomfort, and pulmonary edema. On examination, she was hypertensive (BP 200/140mmHg), tachycardic, and hypoxemic.

Electrocardiography showed sinus rhythm with nonspecific repolarization abnormalities. Transthoracic echocardiography revealed left ventricular (LV) dilation with depressed systolic function (LVEF 39%), moderate functional mitral regurgitation, and mild pericardial effusion. Cardiac biomarkers were markedly elevated (hs-cTnI 4576 pg/mL; NT-proBNP 15502 pg/mL).

Despite initial stabilization, she developed cardiogenic shock 48 hours later due to complete atrioventricular block with ventricular rate <20 bpm, requiring temporary pacing.

Computed tomography pulmonary angiography excluded pulmonary embolism. Cardiac magnetic resonance demonstrated myocardial edema and non-ischemic late gadolinium enhancement involving the inferior and inferoseptal walls.

Autoimmune testing was positive for antinuclear, anti-double stranded DNA, lupus anticoagulant, anticardiolipin, and anti-β2 glycoprotein I antibodies, supporting the diagnosis of SLE with secondary antiphospholipid syndrome. Viral serologies, blood cultures, and coronary angiography excluded infectious and ischemic etiologies.

Intravenous methylprednisolone (1 mg/kg/day) and therapeutic enoxaparin were initiated.

Within 72 hours, sinus rhythm recovered, allowing temporary pacemaker removal, with significant clinical, biochemical, and echocardiographic improvement.

Revaluation echocardiography after one week showed normalization of LV dimensions and recovery of LVEF to 53%.

Endomyocardial biopsy performed six weeks later showed no inflammatory infiltrates, giant cells, granulomas, or infectious agents, likely influenced by prior corticosteroid therapy.

At 8-week follow-up, the patient remained asymptomatic with preserved LVEF.

Discussion

This case highlights lupus myocarditis as a rare inaugural manifestation of SLE presenting with AHF and complete atrioventricular block.

Diagnosis relies on clinical suspicion, multimodality imaging, autoimmune testing, and exclusion of ischemic and infectious causes.

Early immunosuppressive therapy with corticosteroids can lead to rapid electrical, structural, and functional cardiac recovery, improving clinical prognosis.

Whodunit? - A challenging case of acute coronary syndrome

João Pedro Faria; Carla Oliveira Ferreira; Bárbara Rocha; Andreia Sousa; Filipe Vilela; Pedro Azevedo; Catarina Vieira

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

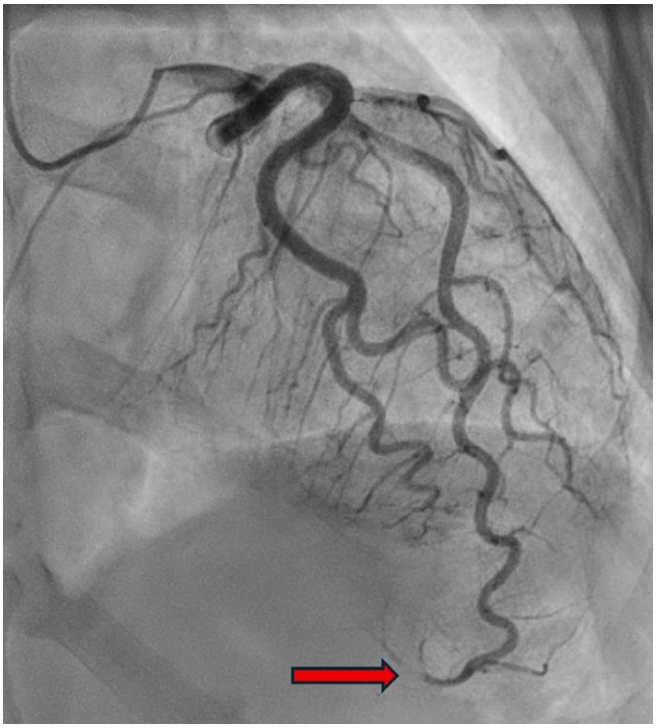
Although most cases of acute coronary syndrome are due to atherosclerotic plaque instability, there are multiple etiologies related to the mismatch between oxygen delivery and demand (type 2 acute myocardial infarction).

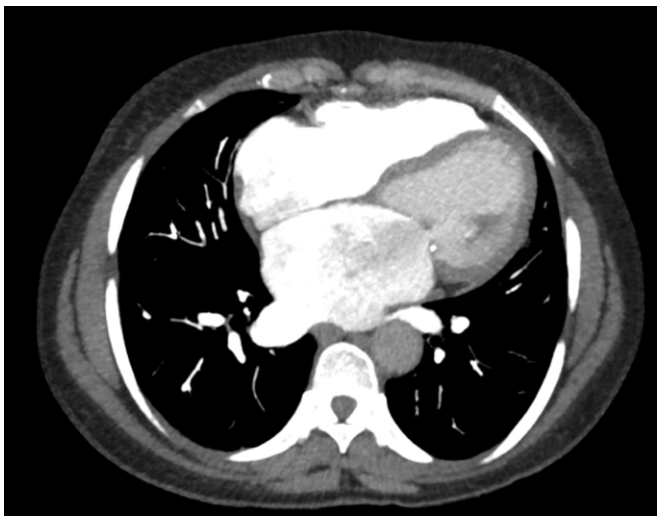
Clinical case

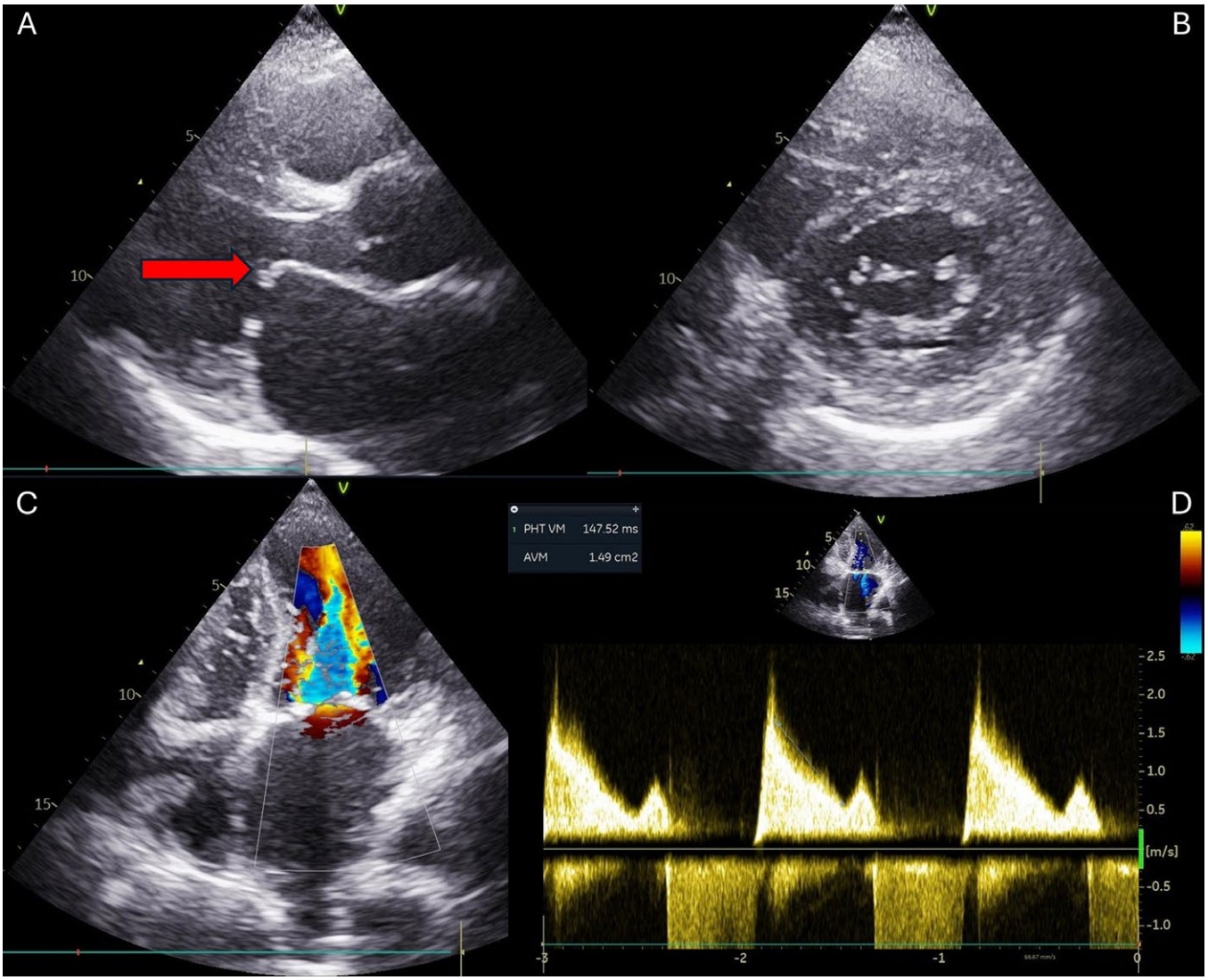
A 39-year-old Black woman with a history of sickle cell disease (SCD), previously hospitalized for vaso-occlusive crisis and treated with hydroxyurea, presented to the Emergency Department with constrictive chest pain radiating to both upper limbs, lasting 12 hours. Physical examination was unremarkable. Electrocardiography showed sinus rhythm without significant abnormalities. Laboratory testing revealed hemoglobin of 9.8 g/dL, peak troponin I of 17 ng/mL and creatine kinase of 390 U/L. Computed tomography pulmonary angiography excluded pulmonary embolism but demonstrated marked left atrial enlargement. Coronary angiography revealed occlusion of the terminal segment of the left anterior descending artery, with a small distal vessel. Given the history of SCD, intensive intravenous hydration and oxygen therapy were initiated. Transthoracic echocardiography demonstrated moderate rheumatic mitral stenosis (mitral valve area of 1.5 cm^2), moderate mitral regurgitation, severe left atrial dilatation, preserved left ventricular systolic function, hypokinesia of the distal interventricular septum and apex, and severe tricuspid regurgitation, with a pulmonary artery systolic pressure of 45 mmHg. During hospitalization, frequent episodes of paroxysmal atrial fibrillation (AF) were documented. Considering the stable hemoglobin levels throughout admission and the absence of significant disease in the remaining coronary arteries, a cardioembolic etiology was assumed, and anticoagulation with warfarin was initiated. The patient evolved favorably and was discharged.

Discussion

In patients with multiple predisposing factors, it can be difficult to identify the most probable etiology and choose the best therapeutic option. Sickle cell disease is associated with a broad spectrum of cardiovascular complications, including myocardial ischemia and pulmonary hypertension. Acute coronary syndrome in SCD is uncommon and may result from arterial vaso-occlusive crisis. Rheumatic mitral stenosis further increases thromboembolic risk, particularly in the presence of AF and severe left atrial enlargement. This case highlights the complex interplay between SCD, rheumatic mitral valve disease, and AF as contributors to myocardial infarction. Although vaso-occlusive ischemia is a recognized mechanism in SCD, the angiographic findings, the severe mitral stenosis and the diagnosis of AF supported a cardioembolic mechanism. Recognition of alternative etiologies of myocardial infarction is essential, as management may differ substantially, particularly regarding anticoagulation and secondary prevention strategies.



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When a STEMI arrives too late

Joana Guerreiro Pereira; Filipa Almeida Saraiva; Alexandru Babiciu; Lucas Hamman; Sofia Andraz; Joana Massa Pereira; Raquel Menezes Fernandes; João Sousa Bispo; Jorge Mimoso

UNIDADE LOCAL DE SAÚDE DO ALGARVE

Introduction

Late presenting ST-elevation myocardial infarction (STEMI) could be associated with several complications. Management becomes especially challenging when associated with cardiogenic shock and recurrent ventricular arrhythmias.

Case Report

A 52-year-old man with arterial hypertension, obesity and heavy smoking habits was admitted with an anterior STEMI with persistent (>24h) symptoms. Coronary angiography revealed severe three-vessel disease, including severe calcified proximal left anterior descending (LAD) disease, mid-LAD occlusion, chronic total occlusions (CTO) of the circumflex and right coronary arteries, and severe ramus intermedius (RI) stenosis. Percutaneous coronary intervention (PCI) of the LAD, diagonal branch and RI was performed.

Hours later, the patient developed cardiogenic shock. Echocardiography showed rapidly enlarging pericardial effusion with cardiac tamponade. Emergency pericardiocentesis drained 450mL of haemorrhagic fluid, with transient haemodynamic recovery. Due to recurrent tamponade, repeat angiography demonstrated distal diagonal branch perforation with active pericardial contrast extravasation, successfully treated with coil embolization.

The patient subsequently developed post-procedural pericarditis and initiated anti-inflammatory treatment. Echocardiography showed a mild reduced left ventricular ejection fraction, with apical akinesia and intracardiac thrombus, requiring anticoagulation. Thirteen days after the index event, recurrent late ventricular fibrillation (VF) episodes occurred, prompting intravenous amiodarone initiation. Despite the absence of clinical evidence of acute ischaemia, repeat angiography revealed LAD re-occlusion and severe diagonal branch stenosis. Complex IVUS-guided revascularization was performed. Ongoing electrical instability led to beta-blocker up-titration, magnesium sulphate and lidocaine infusion. Ablation was considered, but patient improved after combined antiarrhythmic therapy. Further angiographic reassessment excluded new obstructive lesions. A cardioverter-defibrillator was implanted for secondary prevention. The patient was discharged with arrhythmology follow-up.

Discussion

This case highlights the complexity of managing mechanical and electrical complications after a late-presenting STEMI with complex multivessel coronary artery disease. Coronary perforation with haemorrhagic tamponade is a catastrophic PCI complication, requiring prompt recognition and invasive treatment. Recurrent late VF emphasized the arrhythmogenic substrate associated with residual ischaemia, despite clinically silent, and extensive myocardial scar. We underscore the importance of individualized management, clinical suspicion and close rhythm surveillance in patients with late-presenting STEMI.

The heart remembers

João Pedro Faria; Bárbara Rocha; Andreia Sousa; Carla Oliveira Ferreira; Filipe Vilela; Pedro Azevedo; Catarina Vieira

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

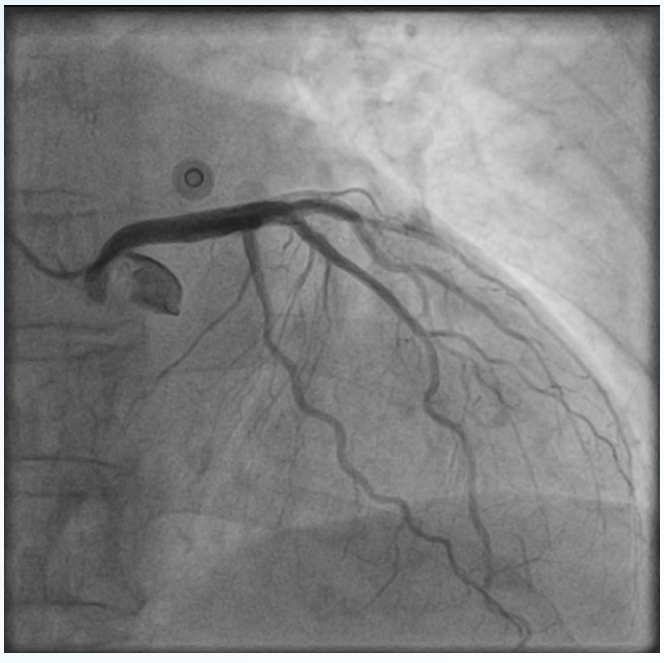
Diffuse T-wave inversion on electrocardiography may have multiple etiologies. Among the most frequent and clinically relevant is myocardial ischemia. However, the differential diagnosis may be broad.

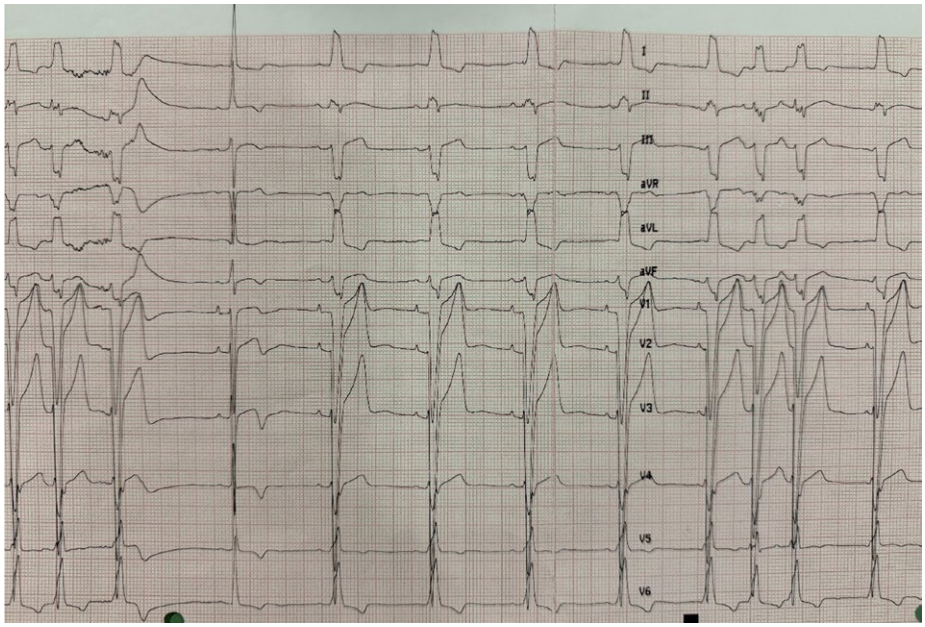
Clinical case

An 81-year-old woman with a history of atrial fibrillation (AF) presented to the Emergency Department with worsening dyspnea and left-sided precordial chest pain. Physical examination revealed coarse bibasal crackles on pulmonary auscultation and a diastolic murmur on cardiac auscultation. Electrocardiography showed sinus rhythm, left bundle branch block (LBBB), and frequent supraventricular premature beats. She was initially diagnosed with acute decompensated heart failure. Intravenous furosemide was started, and she was admitted for further management. Transthoracic echocardiography demonstrated moderate left ventricular dilatation with eccentric hypertrophy, severely reduced systolic function, moderate-to-severe aortic regurgitation, and moderate mitral regurgitation. During hospitalization, frequent episodes of AF with intermittent LBBB were documented. When QRS morphology was normal, biphasic T waves were observed in leads V1-V4. Given these electrocardiographic changes, the severe left ventricular systolic dysfunction and the chest pain complaints, coronary angiography was performed and excluded epicardial coronary artery disease. To further clarify the severity of aortic regurgitation, transesophageal echocardiography was performed, confirming left ventricular dilatation and severe systolic dysfunction, with moderate aortic regurgitation (vena contracta 5 mm, effective regurgitant orifice area 0.12 cm^2, regurgitant volume 44 mL). Amiodarone therapy was initiated, achieving adequate rhythm control, and heart failure symptoms resolved with continued diuretic therapy. The patient showed favorable clinical evolution and was discharged, with subsequent outpatient Cardiology follow-up.

Discussion

Cardiac memory T waves are persistent T-wave changes occurring after restoration of normal ventricular activation following a period of wide QRS rhythm, such as LBBB, ventricular tachycardia, or ventricular pacing. Although they represent an adaptive response to abnormal ventricular depolarization, they are frequently misinterpreted as other pathological entities, particularly Wellens syndrome, which is characterized by biphasic or inverted T waves in leads I, aVL, and in precordial leads. In contrast to the Wellens pattern, cardiac memory T waves are identified when T-wave polarity follows the polarity of the QRS complexes during aberrant conduction. In memory T waves after LBBB, the QRS complex is typically positive in leads I and aVL and negative in the precordial leads. This phenomenon should therefore be considered in the differential diagnosis of patients presenting with diffuse T-wave inversion.

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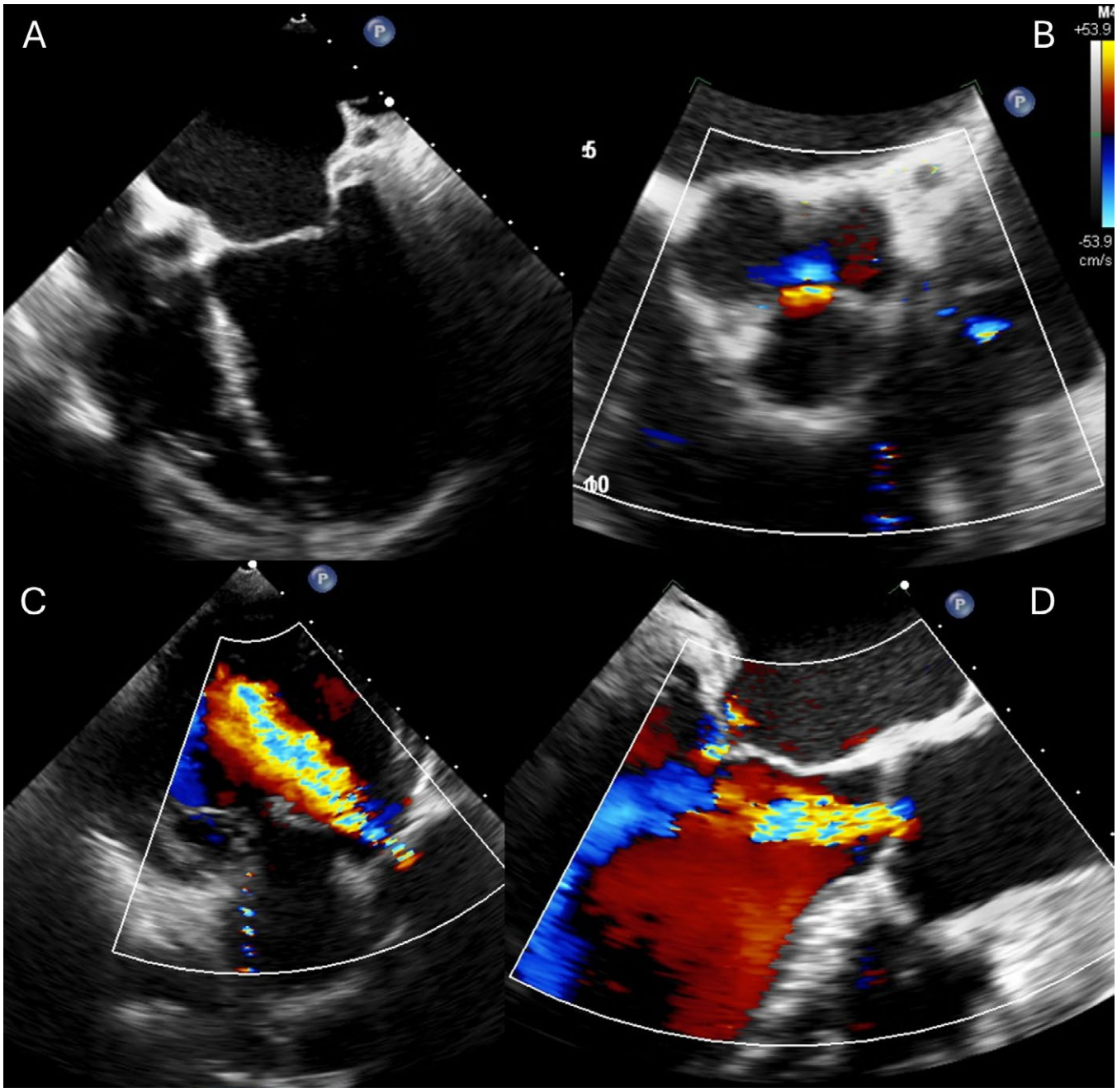
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Behind the rhythm - a not so innocent case of atrial fibrillation

João Pedro Faria; Bárbara Rocha; Andreia Sousa; Carla Oliveira Ferreira; Filipe Vilela; Rui Files Flores; Catarina Vieira

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

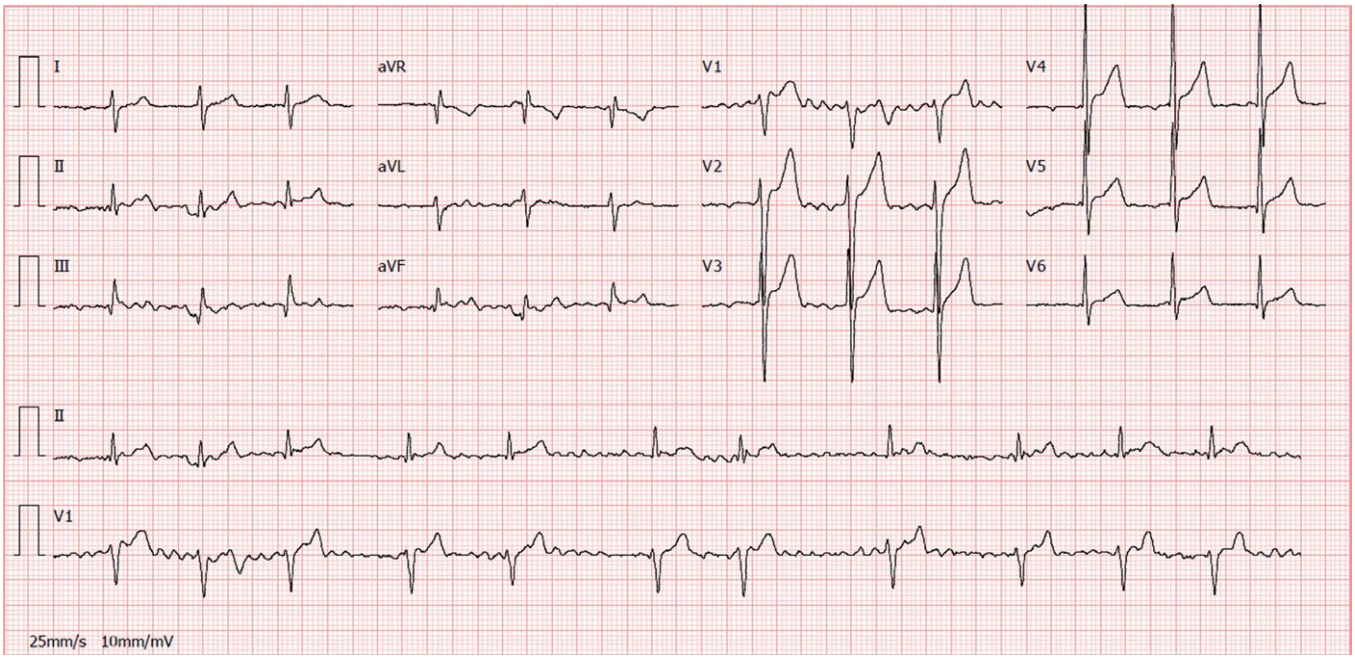
Atrial fibrillation (AF) is the most common atrial tachyarrhythmia, predominantly affecting older individuals and patients with cardiovascular risk factors. However, AF may also occur as an early manifestation of an underlying cardiomyopathy, particularly in the presence of genetic substrates associated with myocardial fibrosis and arrhythmic risk.

Clinical case

A 49-year-old man was referred to Cardiology consultation due to paroxysmal AF. Twelve years earlier, he had been evaluated for the same condition, and transthoracic echocardiography (TTE) revealed moderate left ventricular systolic dysfunction. Following successful electrical cardioversion to sinus rhythm, his systolic function recovered and the patient was lost to follow-up. In the next 12 years, he experienced multiple AF recurrences. Despite pulmonary vein isolation, recurrent AF episodes were later documented. Repeat TTE demonstrated mild left ventricular systolic dysfunction with global hypokinesia, more pronounced in the lateral wall and basal-to-mid posterior segments, and his resting ECG revealed inferior Q waves; a coronary CT angiography excluded coronary artery disease. A cardiac magnetic resonance revealed extensive subepicardial late gadolinium enhancement involving the inferior, inferolateral, and anterolateral walls, with preserved left ventricular systolic function, which were initially considered as sequelae of a previous acute myocarditis. He denied a family history of sudden cardiac death, although he had limited contact with close relatives. During follow-up, the patient reported that his mother had been diagnosed with a FLNC gene mutation (c.3937C>T variant), and genetic testing confirmed the presence of the same mutation in the patient. He remains under regular follow-up and is currently being considered for implantation of a loop recorder.

Discussion

Filamin C (FLNC) gene mutations have emerged as important causes of inherited cardiomyopathies, frequently associated with myocardial fibrosis, ventricular dysfunction, and increased arrhythmic risk. Although ventricular arrhythmias are usually emphasized, atrial arrhythmias may represent an early clinical manifestation, preceding overt structural disease. In this case, recurrent AF in a relatively young patient, associated with unexplained left ventricular dysfunction and extensive non-ischemic fibrosis on cardiac magnetic resonance imaging, raised suspicion for an underlying cardiomyopathy. The identification of a pathogenic FLNC variant established the diagnosis. This case highlights the importance of comprehensive etiological investigation in younger patients with AF and subtle structural abnormalities. Early recognition of inherited cardiomyopathies allows tailored surveillance, family screening, and appropriate risk stratification for malignant ventricular arrhythmias.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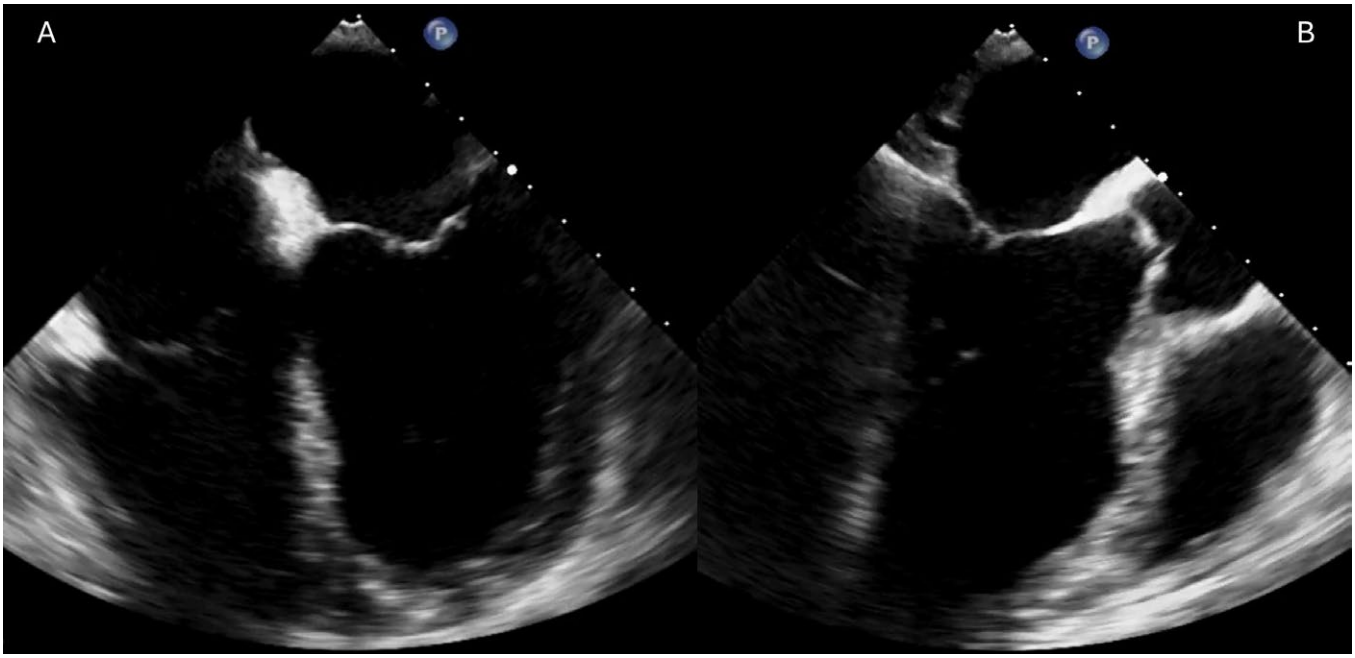
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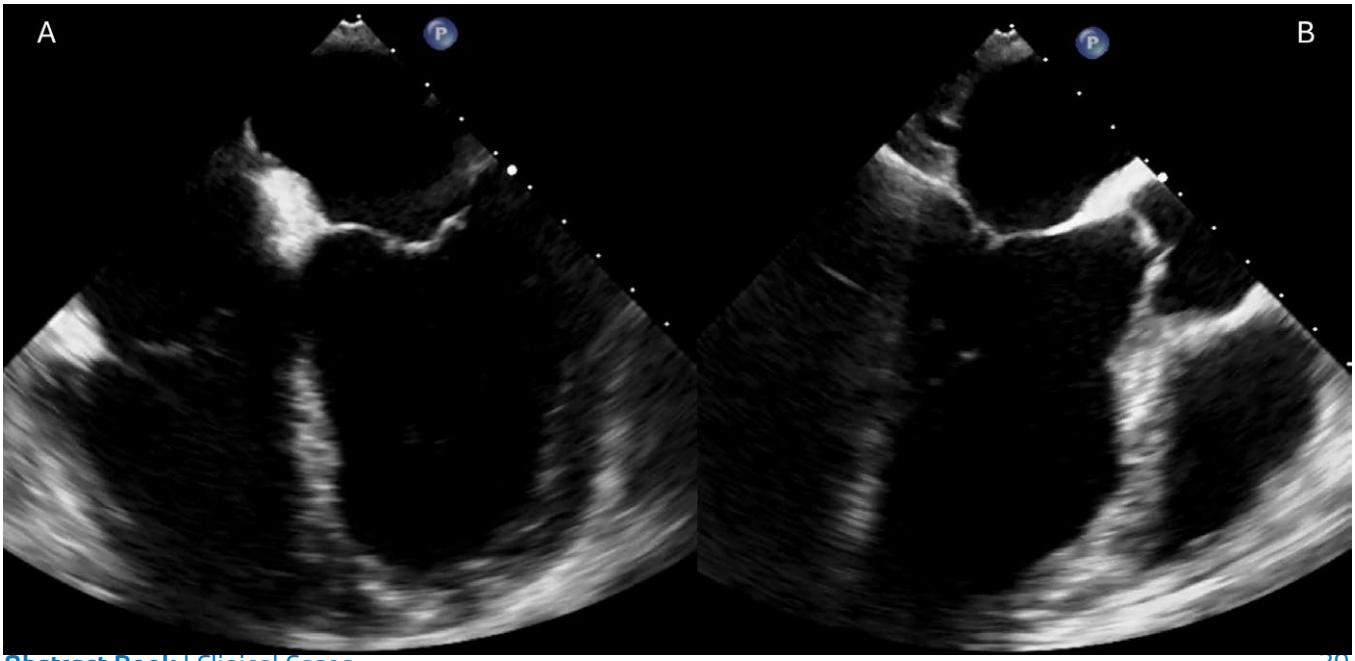
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From Abdominal Pain to Stroke: A Challenging Case of Infective Endocarditis

Filipa Almeida Saraiva; Alexandru Babiciu; Joana Guerreiro Pereira; Lucas Hamann; Sofia Andraz; Joana Massa Pereira; Dina Bento; Raquel Menezes Fernandes; Jorge Mimoso

UNIDADE LOCAL DE SAÚDE DO ALGARVE

Introduction

Infective endocarditis (IE) remains a diagnostic challenge due to its variable clinical presentation. Early recognition is essential due to its high morbidity and mortality.

Clinical Case

A 29-year-old man with no significant medical history or drug use presented to the emergency department with a 3-day history of abdominal pain. Laboratory findings showed leukocytosis (14.0 × 10?/L) with neutrophilia (11.5 × 10?/L) and elevated C-reactive protein (69 mg/L, ref <5 mg/L).

He was discharged with analgesic therapy but returned 6 hours later due to new onset of speech disturbances. Blood pressure and heart rate were normal; he was subfebrile (T 37.8ºC). Physical examination revealed an holosystolic murmur at the apex and pain at left abdominal quadrants. Neurological examination revealed moderate aphasia and right homonymous hemianopsia.

Contrast-enhanced CT imaging showed a left temporal hypodensity, consistent with an ischemic stroke in the left middle cerebral artery territory. As the infarction was already established, thrombolysis was not indicated. Thoracoabdominal CT further revealed splenic infarction and a mycotic aneurysm of the superior mesenteric artery.

Electrocardiography on admission showed sinus rhythm at 86 bpm, poor R-wave progression and peaked T waves.

Due to infarctions in multiple vascular territories, a transthoracic echocardiogram was performed, revealing large vegetations on both mitral valve leaflets. Transesophageal echocardiography provided further characterization, showing a 14 × 6 mm vegetation on the anterior leaflet and two vegetations (10 × 7 mm and 13.8 × 5 mm) on the posterior leaflet, complicated by leaflet perforation and moderate regurgitation.

The patient was admitted with the diagnosis of complicated IE involving the mitral valve.

He completed 7 days of empirical antibiotic therapy with ampicillin, ceftriaxone and gentamicin. Blood cultures isolated *Streptococcus oralis* sensitive to penicillin G and cefotaxime, leading to de-escalation of antibiotic therapy to ceftriaxone. Repeated blood cultures were negative.

He was submitted to mitral valve replacement and to subsequent embolization of the superior mesenteric artery aneurysm. The patient underwent a rehabilitation program including physiotherapy and speech therapy, with improvement of aphasia. He was later admitted to the Home Hospitalization to complete ceftriaxone therapy, which was continued for 4 weeks after negative blood cultures.

Discussion

This case illustrates a severe presentation of IE, with large vegetations, leaflet perforation and systemic embolization in a previously healthy young man. Early diagnosis and multidisciplinary approach were crucial for a favourable outcome.

We emphasize the importance of suspecting IE in patients presenting with embolic events affecting multiple vascular territories, event at a young age and without predisposing conditions.

The hypertrophic enigma

João Pedro Faria; Bárbara Rocha; Andreia Sousa; Carla Oliveira Ferreira; Filipe Vilela; Catarina Vieira

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

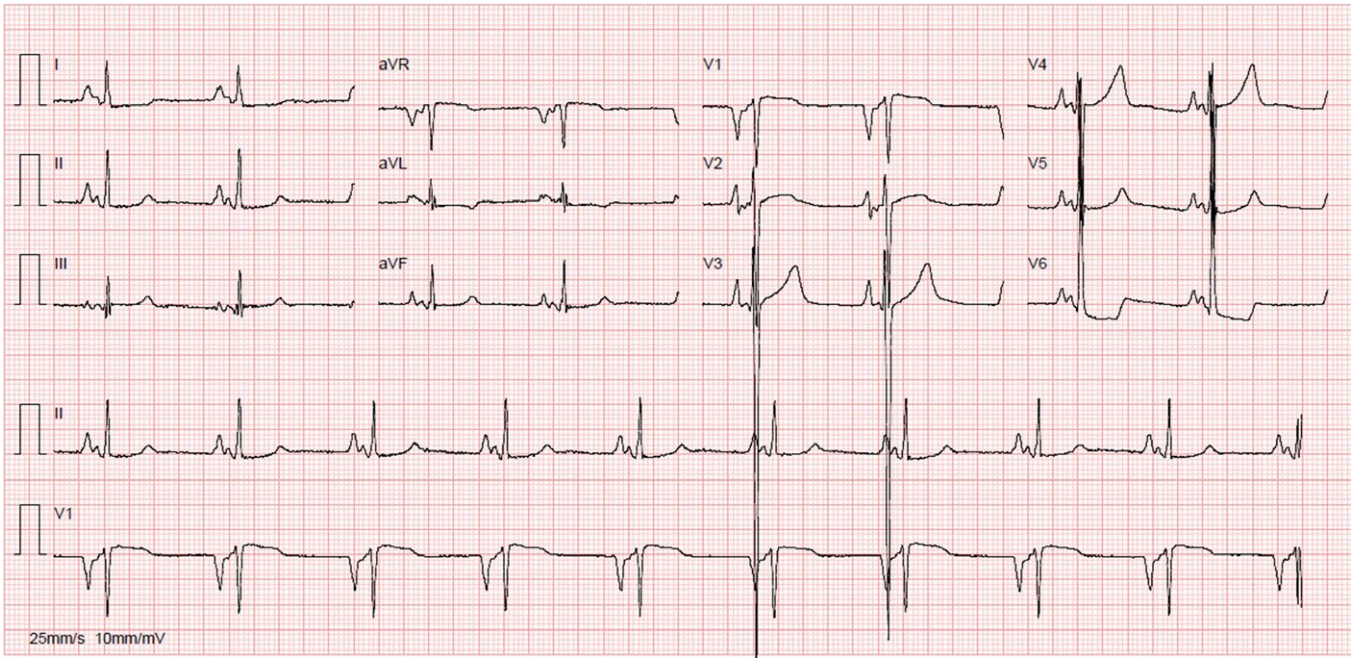
Hypertrophic cardiomyopathy (HCM) is a genetically heterogeneous disease characterized by unexplained left ventricular hypertrophy (LVH) and variable clinical presentation. Distinguishing HCM from other cardiomyopathies may be challenging in young patients with atypical findings or systemic abnormalities.

Clinical Case

A 22-year-old Pakistani man with beta-thalassemia minor was referred to Cardiology after transthoracic echocardiography, performed due to exertional fatigue, revealed biatrial dilatation, severe LVH with infiltrative appearance and preserved left ventricular systolic function, with a resting intraventricular gradient of 43 mmHg. He had a sister with a heart disease he could not specify. On physical examination, he was emaciated and pale. His ECG showed sinus rhythm, left atrial enlargement and LVH with strain pattern. Laboratory testing revealed eosinophilia. Nebivolol, furosemide and albendazole were initiated, with no significant clinical improvement, but with resolution of eosinophilia. Cardiac magnetic resonance (CMR) demonstrated normal-sized ventricles with preserved systolic function, asymmetric LVH, involving the mid interventricular septum and the basal-to-mid anterior, lateral, and inferior walls, with a maximum thickness of 19 mm in the mid-anterior segment and a more apical insertion of the papillary muscles. Tissue characterization revealed septal fibrotic striae and extensive diffuse fibrosis in the most hypertrophied segments. Native T1 mapping was 960 ms and T2* was 42 ms. Genetic testing identified the c.1051A>G variant in the *LDB3* gene in heterozygosity and the c.439G>C variant in the *TNNI3* gene in homozygosity, both of uncertain significance. These findings were consistent with HCM, and his HCM Risk Score was 3.5%. Despite a normal Holter exam, and due to the diffuse fibrosis, the patient was proposed for implantation of a cardioverter-defibrillator, which he refused. However, he died unexpectedly shortly after, with no definitive cause of death established.

Discussion

This case highlights the diagnostic complexity of HCM. The patient's clinical profile, ECG and imaging characteristics initially raised suspicion for infiltrative myocardial disease. Additionally, beta-thalassemia led to consider secondary cardiomyopathies, such as iron-overload disease. Nevertheless, extensive investigation supported obstructive HCM with an early, aggressive phenotype. CMR was critical for diagnosis, providing findings consistent with HCM. Normal T1 and T2* values argued against infiltrative or iron-overload cardiomyopathy. Although genetic variants were classified as of uncertain significance, their association with cardiomyopathic phenotypes reinforced the hypothesis of primary HCM. The extensive fibrosis likely acted as an arrhythmogenic substrate, potentially explaining the patient's sudden death.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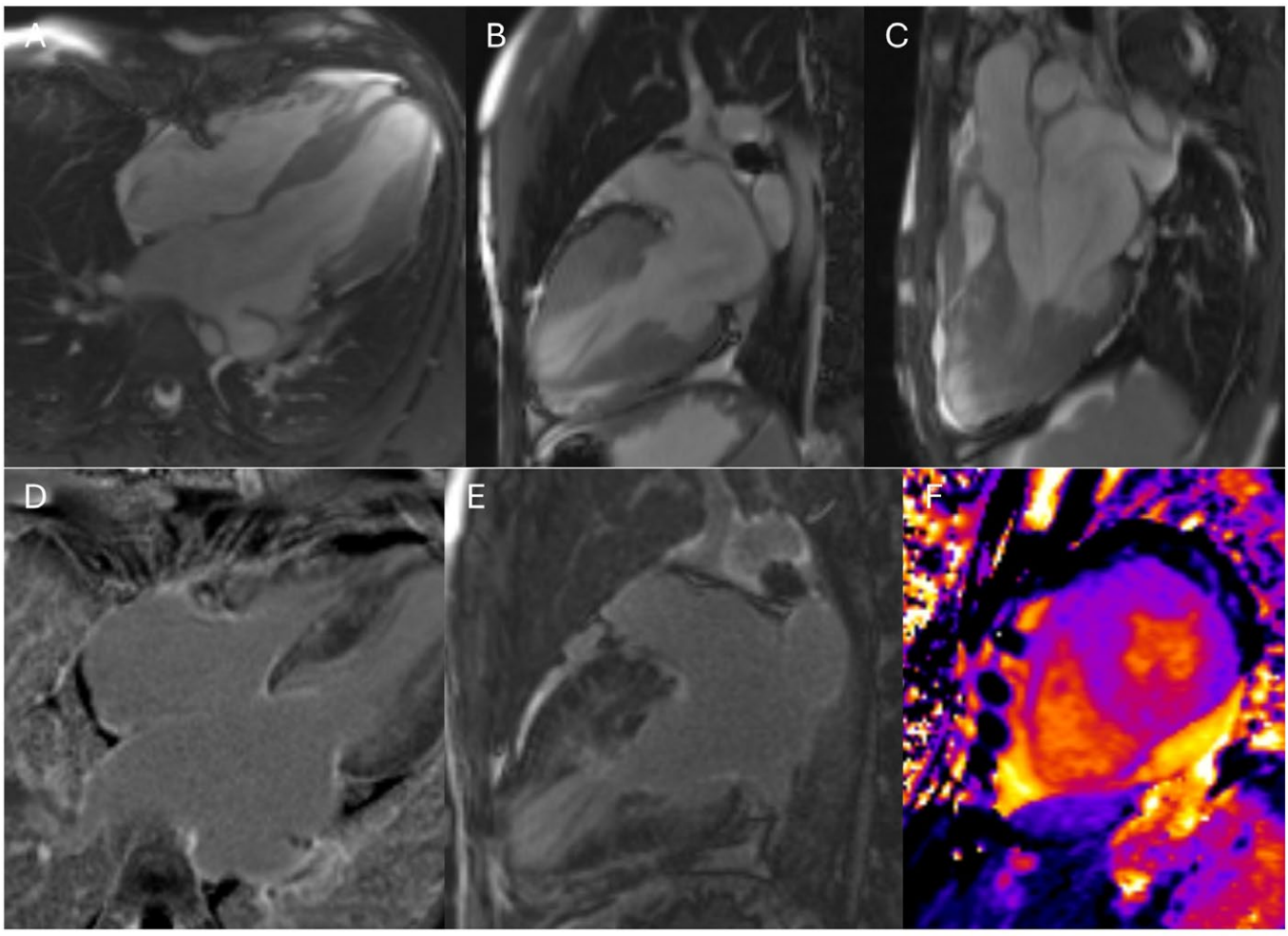
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A case of early referral for advanced heart failure treatment

David Campos; Patrícia Bernardes; Jéni Quintal; Marco Tomaz; Ivo Palmeiro; Leonel Pereira; Tatiana Duarte; Filipe Seixo; Sara Gonçalves

UNIDADE LOCAL DE SAÚDE DA ARRÁBIDA

Introduction

The prevalence of advanced heart failure is increasing, while prognosis remains poor, with many patients often referred to Advanced Heart Failure Centres too late. An adequate and timely identification of warning signs is needed for an early approach and outcome improvement.

Case Report

We present a male, 62 years old, with a personal history of dilated miocardiopathy with an initial ejection fraction (EF) of 24%, followed up by our Heart Failure Centre since 2020. CRT-D was placed at 08/2020 due to persistent severe EF reduction and left bundle branch block. Clinically stable since then until 04/2025, when the patient presents with worsening dyspnea, now at daily life activities, orthopnea, and symptomatic hypotension, with a reduction of EF to 15%. Unable to tolerate sacubitril fue to hypotension, the patient started levosimendan cycles biweekly and was refered to an Advanced Heart Failure Centre. With an INTERMACS score of 4-5, left ventricular assisting device (LVAD) and heart transplantation (HT) were option treatments discussed.

At 12/10/2025, the patient starts with worsening dyspnea, fatigue and hypotension. Laboratory studies revealed hyperlactatemia, acute renal failure KDIGO I, and worsening chronic liver failure. Echocardiogram at bedside revealed an EF of 10-15%. Admitted at the ICU with cardiogenic shock SCAI D, furosemide, dobutamine and levosimendan perfusions were started, with clinical and laboratory improvement. The case was discussed with the Advanced Heart Failure Centre mentioned above, being decided that LVAD would be the best immediate strategy. The patient was then transfered for implantation of HEARTMATE 3. The procedure went without complications, with a steady and progressive improvement of congestion. Finally, after pharmacological optimization and physical rehabilitation, the patient was discharged 1 month after LVAD placement, with no symptoms or signs of congestion or hypoperfusion. Follow up appointments proved clinical stability, with a NYHA I presentation at the last appointment, April of 2026, while waiting for HT.

Discussion

This case report demonstrates that early identification and referral of advanced heart failure is important for its adequate treatment. CRT non responder, ARNI intolerance, EF < 25 % and an INTERMACS of 4-5 were all warning signs for an early referral to LVAD or HT. Finally, cardiogenic shock with worsening renal and hepatic function further reinforces the need for an immediate approach. Although HT remains the gold standard treatment, organ donor shortage is a main limitation. Therefore, LVAD remains as na alternative option, usually as a bridge to candidacy or to transplantation. In this scenario, the HERATMATE 3 led to a significant clinical improvement, allowing the patient to live longer and with quality of life, while awaiting for a more definite approach.

Beyond the ST Elevation: An Acute Type A Aortic Dissection Presenting as Myocardial Infarction

Filipa Almeida Saraiva; Alexandru Babiciu; Joana Guerreiro Pereira; Lucas Hamann; Sofia Andraz; Joana Massa Pereira; Dina Bento; Raquel Menezes Fernandes; Jorge Mimoso

UNIDADE LOCAL DE SAÚDE DO ALGARVE

ST-segment elevation myocardial infarction (STEMI) is usually caused by acute coronary thrombosis over a ruptured atherosclerotic plaque. However, ST-segment elevation may also result from other potentially life-threatening conditions that mimic acute coronary syndrome. Prompt diagnosis is crucial for early treatment

Clinical Case

A 79-year-old man without known cardiovascular risk factors, presented to the emergency department with sudden severe abdominal pain radiating to the back and lower limbs. He was hypotensive (BP 65/54 mmHg), bradycardic (HR 40 bpm), tachypneic (RR 26 cpm), SpO2 100%. Cardiopulmonary auscultation revealed no audible murmurs or abnormal breath sounds. Arterial blood gas analysis revealed respiratory alkalosis and hyperlactacidemia (lactate 3.5 mmol/L). Laboratory tests showed elevated transaminases (AST 684 UI/L, ALT 660 UI/L), acute kidney injury (creatinine 1.6mg/dL), elevated troponin-T (52.3 pg/mL).

Electrocardiogram showed bradycardia (HR 40 bpm), significant ST-segment elevation in the inferior leads and ST-segment depression in the remaining ones.

Transthoracic echocardiography showed a left ventricle ejection fraction of ~50% with akinesia of the inferior and posterior walls, right ventricular systolic dysfunction and moderate aortic regurgitation.

Assuming the diagnosis of STEMI, the patient underwent a coronary angiography that revealed a subocclusive stenosis of the proximal right coronary artery. A first attempt to perform angioplasty was made, but the intravascular ultrasound showed an aortic dissection involving the right coronary artery, leading to an immediate interruption of the procedure. It was also noted that there were signs of contrast retention in the ascending aorta.

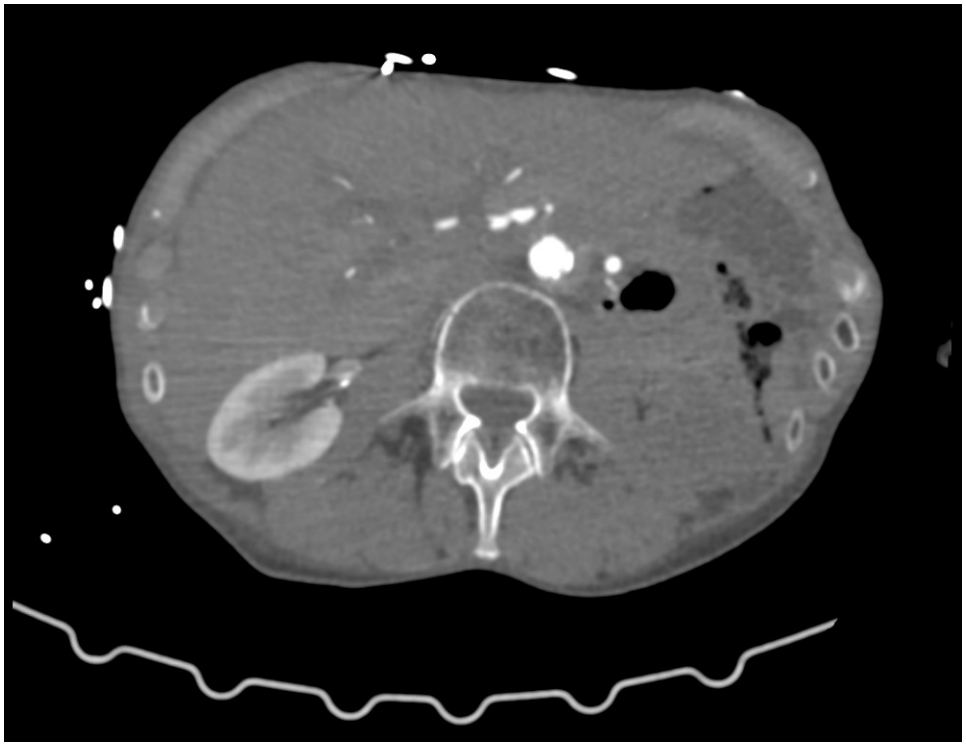
Computed tomography angiography of the thorax, abdomen and pelvis revealed an extensive Stanford type A aortic dissection extending to all four supra-aortic vessels, left renal artery leading to renal hypoperfusion and occlusion of the right superficial femoral artery.

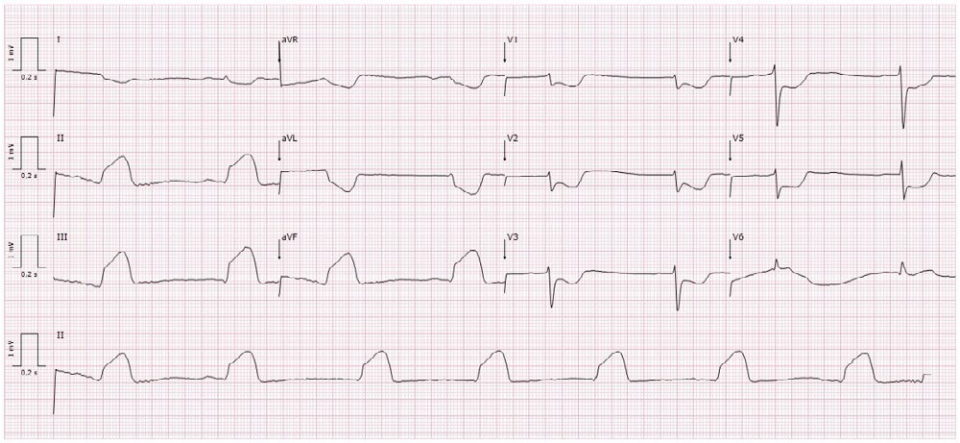
The patient was stabilized and transferred to a tertiary hospital for emergency surgery. Upon arrival, he was in cardiorespiratory arrest. Advanced life support was initiated but return of spontaneous circulation was not achieved and the patient did not survive.

Discussion

Stanford type A aortic dissection is a highly lethal condition that may mimic a myocardial infarction, particularly when the coronary ostia are involved. This clinical and electrocardiographic overlap may delay the diagnosis and referral for emergency surgery, thereby increasing even more the likelihood of an adverse outcome.

The initial presentation of this patient was consistent with an inferior STEMI. The presence of severe abdominal pain radiating to the back, hypotension and acute aortic regurgitation, should emphasize the importance of differential diagnosis, particularly acute aortic dissection.

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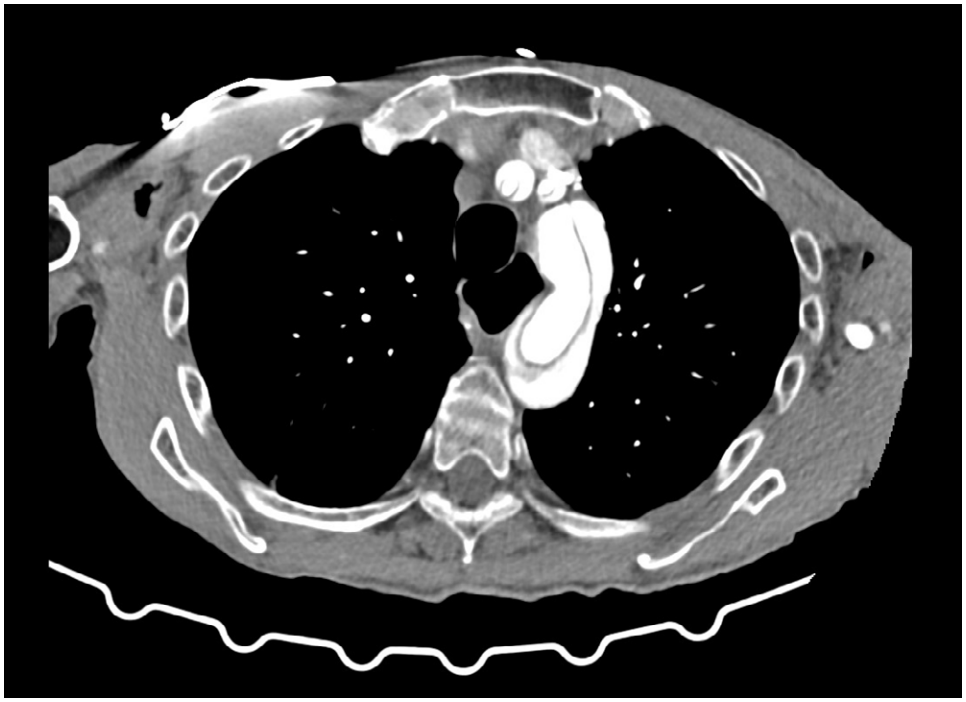


Syncope: Blame the Wasp?

Joana Guerreiro Pereira; Alexandru Babiciu; Filipa Almeida Saraiva; Lucas Hamman; Sofia Andraz; Joana Massa Pereira; Raquel Menezes Fernandes; Pedro de Azevedo; Jorge Mimoso

UNIDADE LOCAL DE SAÚDE DO ALGARVE

Introduction

Syncope may pose a diagnostic challenge when multiple potential aetiologies coexist. Although most episodes are benign, identifying a cardiac cause is crucial given its association with increased morbidity and mortality, to allow for timely treatment.

Case Description

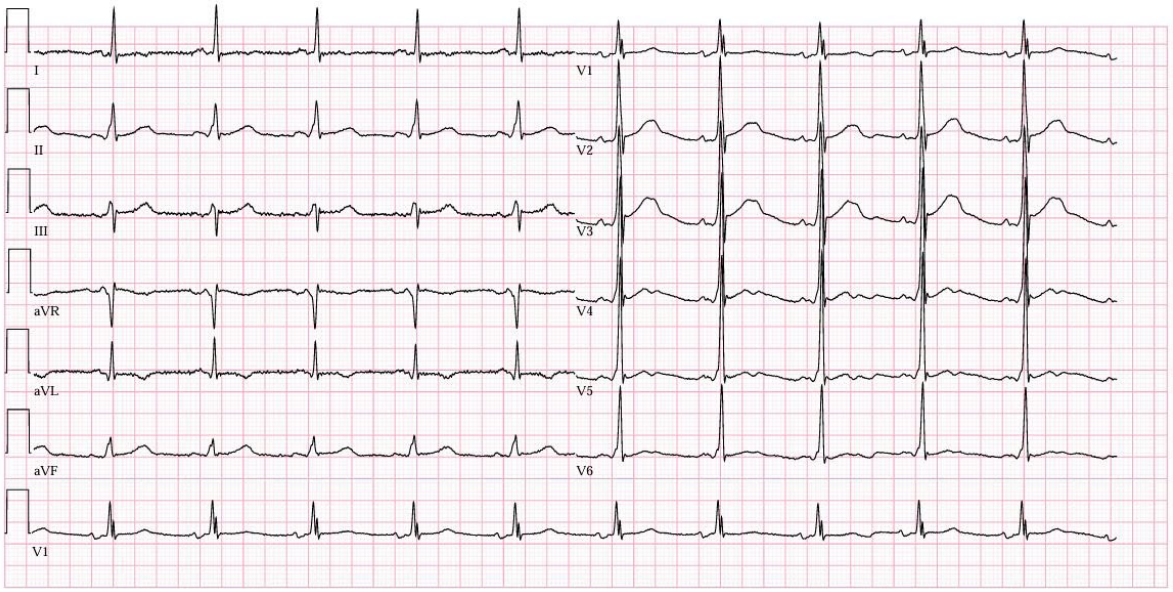
A 61-year-old man with dyslipidaemia was admitted in the Emergency Department (ED) after a wasp sting followed by a transient loss of consciousness associated with urinary incontinence. He did not recall having prodromal symptoms. Recovery was spontaneous and complete, without neurological deficits. The patient denied any previous episodes of syncope. He remained haemodynamically stable throughout evaluation, without objective signs of anaphylaxis.

The electrocardiogram showed sinus rhythm and manifest ventricular pre-excitation suggestive of a left-sided accessory pathway (Figure 1). Thorough review of previous records revealed a documented episode of pre-excited atrial fibrillation with rapid ventricular response during a prior ED admission (Figures 2 and 3), together with a history of recurrent palpitations. Despite the temporal association with the wasp sting, these findings raised suspicion of an arrhythmic mechanism. Continuous monitoring showed persistent ventricular pre-excitation without documented arrhythmias. Echocardiography revealed no structural heart disease. The patient was subsequently referred for electrophysiological evaluation.

Electrophysiological study demonstrated a manifest left lateral accessory pathway with an antegrade effective refractory period of 230 ms, indicating the capacity for rapid ventricular conduction. Radiofrequency catheter ablation was successfully performed through a transseptal approach, achieving complete elimination of accessory pathway conduction without inducible arrhythmias at the end of the procedure. The patient will maintain follow-up in the Arrhythmology Consultation.

Discussion

This case illustrates how a plausible alternative diagnosis may divert attention from a potentially high-risk arrhythmic substrate. Although the syncopal episode occurred immediately after a wasp sting, the absence of objective evidence of anaphylaxis and the discovery of previously documented pre-excited atrial fibrillation shifted the diagnostic focus towards a pre-excitation syndrome, leading to the identification of a high-risk accessory pathway. This case emphasises the importance of maintaining a high index of suspicion for arrhythmic causes of syncope, even when alternative explanations appear plausible.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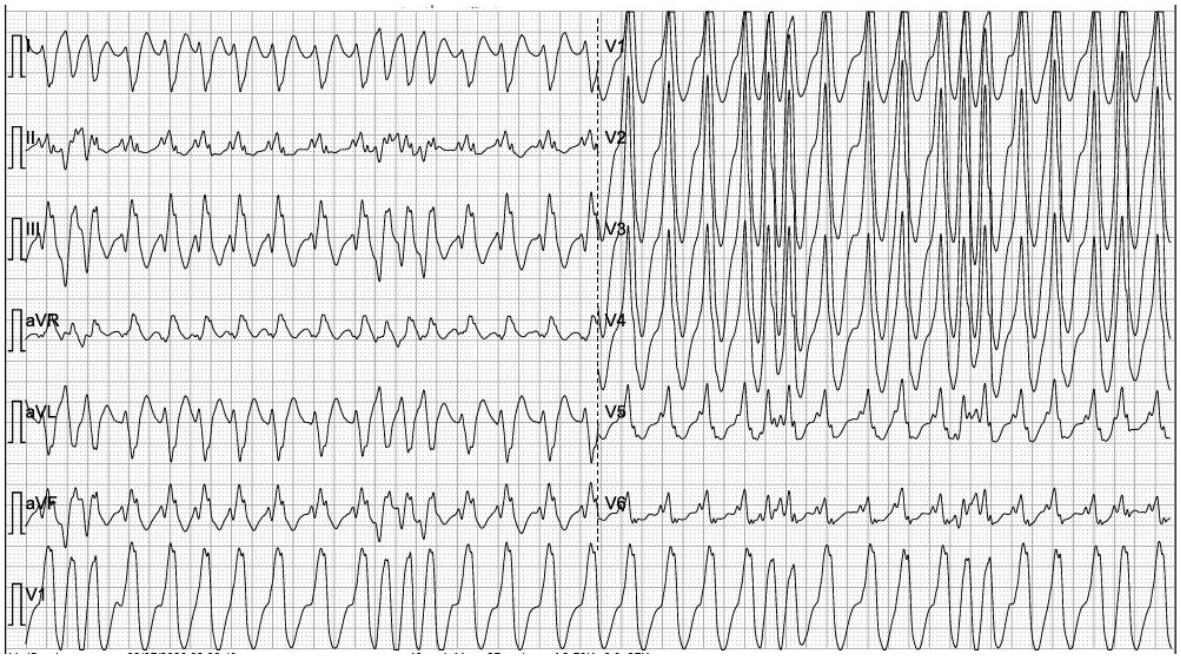
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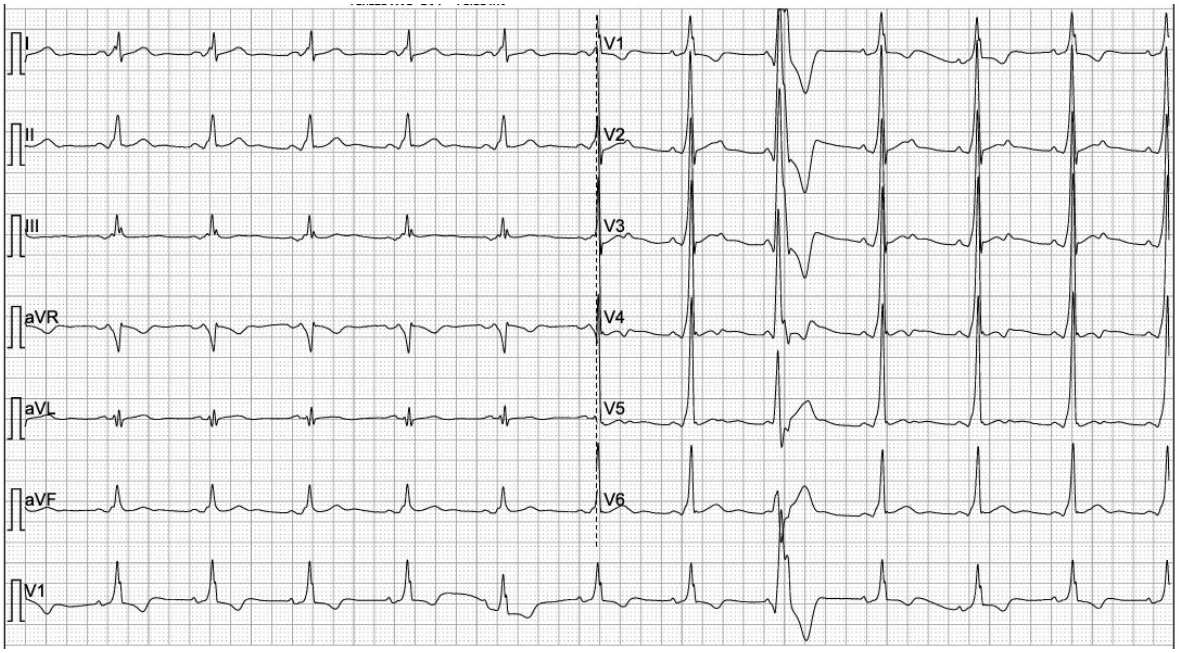
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Superdominant but Vulnerable: The Perils of Ostial Left Anterior Descendent Stenosis in Rare Coronary Anatomy

Bárbara Antunes Rocha; João Faria; Andreia Sousa; Carla Ferreira; Filipe Vilela; Mónica Dias; Sofia Fernandes; Carlos Galvão Braga

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

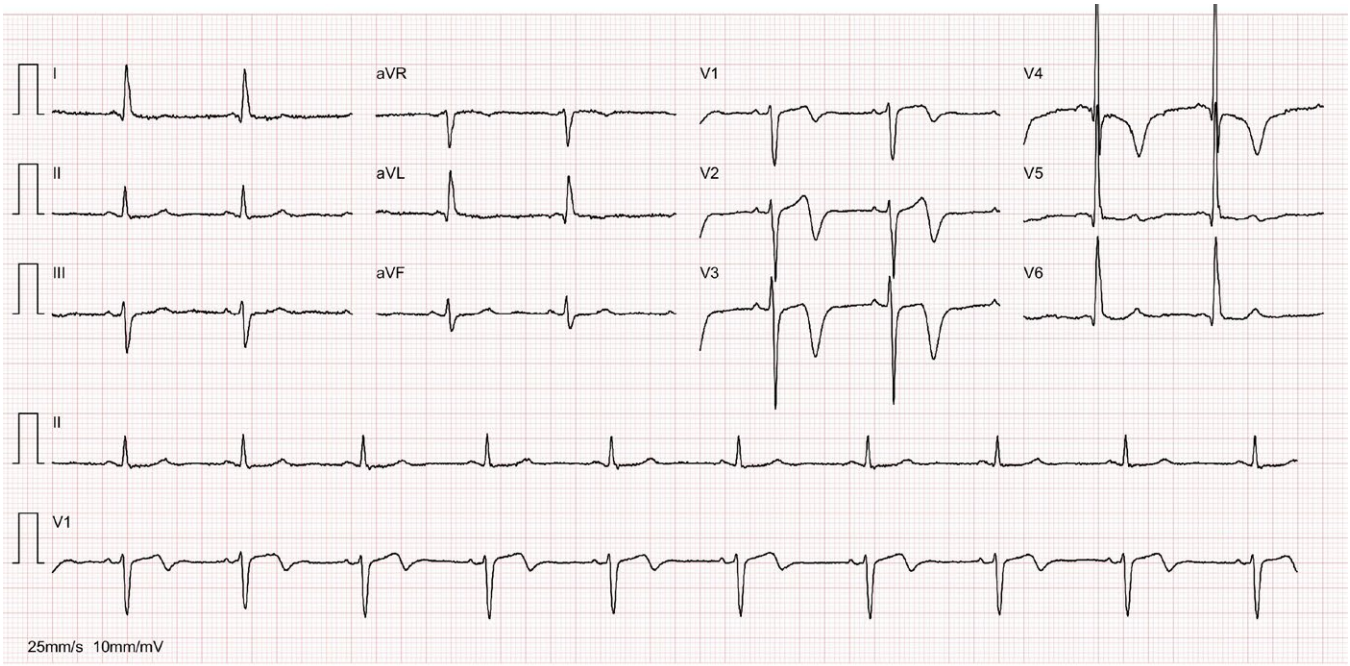
Variations in coronary dominance can markedly influence myocardial perfusion patterns and have significant implications for clinical management. A superdominant circumflex artery, defined as a circumflex artery that supplies not only the posterior descending artery but also the myocardial territory perfused by the right coronary artery, often in the absence of the latter, represents a rare anatomical variant, reported in isolated case studies and estimated to occur in <0.1% of the population. The coexistence of a superdominant circumflex with severe ostial left anterior descending (LAD) stenosis constitutes a challenging scenario for percutaneous coronary intervention due to the risk of myocardial ischemia.

Case report

A 78-year-old male with a history of hypertension and dyslipidemia presented to the emergency department with chest pain, described as oppressive, radiating to the left upper limb, and associated with diaphoresis. On admission, he was hemodynamically stable. Electrocardiography revealed sinus rhythm with T-wave inversion in anterior leads. Laboratory testing demonstrated no anemia or leukocytosis, preserved renal function, and no electrolyte abnormalities. Troponin I was elevated at 0.15 ng/mL, while C-reactive protein remained within normal limits. Transthoracic echocardiography demonstrated preserved biventricular systolic function without segmental wall motion abnormalities. Coronary angiography revealed a calcified 90% ostial LAD stenosis with preserved distal vessel patency. The circumflex artery was identified as superdominant, supplying the posterior descending and posterolateral branches and thereby perfusing the entire myocardial territory normally dependent on the right coronary artery. The right coronary artery was angiographically absent. Given the critical proximal LAD lesion in the context of this unusual anatomy, the case was discussed in heart team. Surgical myocardial revascularization was recommended and accepted by the patient. During hospitalization, he remained asymptomatic (Killip class I), with a peak troponin I level of 1.9 ng/mL. He underwent coronary artery bypass grafting (CABG) without perioperative complications.

Conclusion

This case highlights the clinical relevance of coronary anomalies in risk stratification and revascularization planning. In the presence of a superdominant circumflex, any compromise of left coronary inflow threatens an exceptionally large myocardial mass. Consequently, CABG was considered essential to ensure myocardial perfusion and mitigate the risk of ischemic events. Recognition of such anatomical variants during angiographic evaluation is critical for procedural decision-making and optimizing patient outcomes. This case underscores the importance of tailoring surgical strategies to anatomically complex presentations of coronary artery disease.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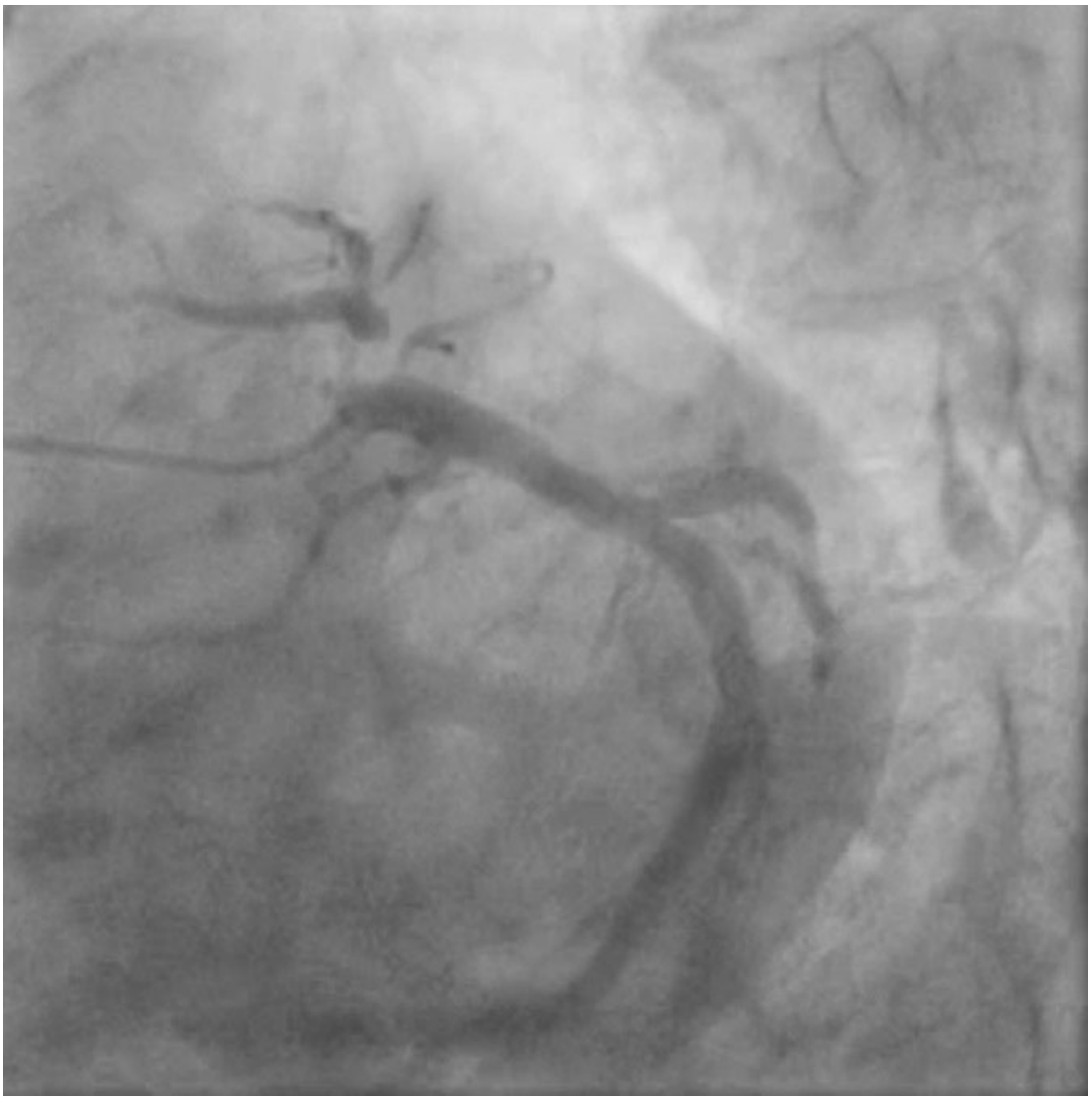
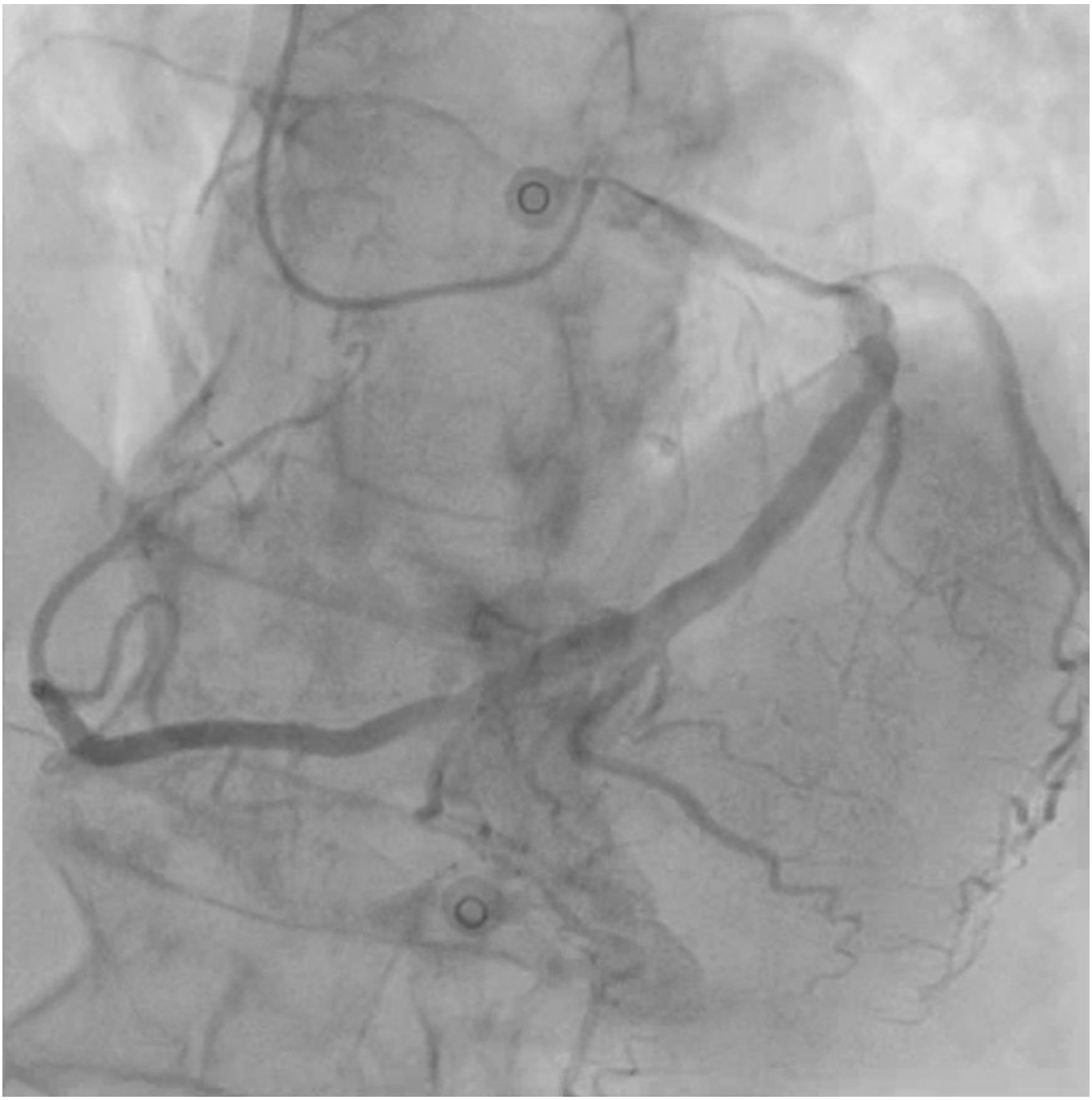




Short-coupled polymorphic ventricular tachycardia as a cause of unexplained syncope

Adriana Vazão; André Martins; Mónica Amado; Joana Reis Pereira; Carolina Esteves; Maksym Baburko; João Carvalho; Hélia Martins; David Durão

UNIDADE LOCAL DE SAÚDE DA REGIÃO DE LEIRIA

Introduction

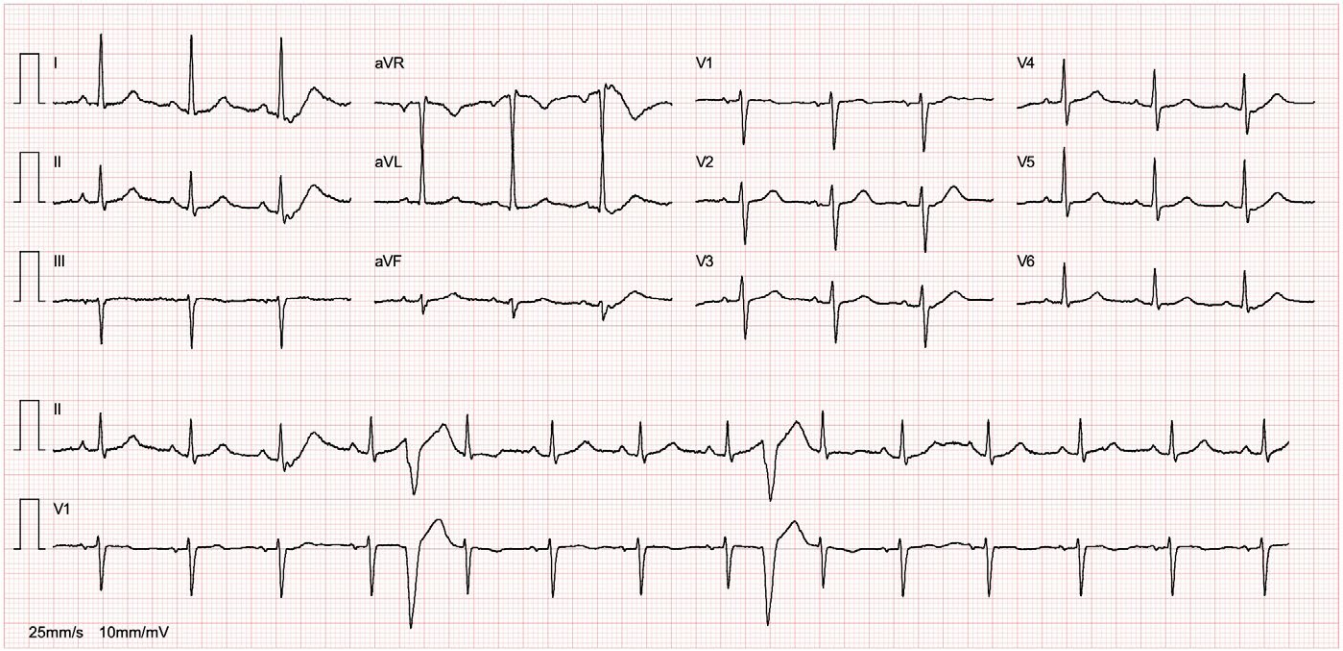
Short-coupled polymorphic ventricular tachycardia (PVT) is a rare but potentially malignant arrhythmic syndrome characterized by ventricular arrhythmias (VA) triggered by short-coupled premature ventricular complexes (PVCs) in patients without structural heart disease or long QT syndrome. Early recognition is crucial given the risk of sudden cardiac death (SCD).

Case description

A 54-year-old woman was referred for cardiology evaluation after abnormalities detected on 24-hour Holter monitoring requested following two syncopal episodes. Her medical history included hypertension treated with atenolol, without family history of SCD. The first syncopal episode occurred while seated in a car and was associated with abnormal upper-limb movements and ocular deviation. Five months later, she experienced a second abrupt syncopal episode while gardening, without prodromes and followed by spontaneous recovery. Baseline electrocardiogram showed sinus rhythm with PVCs displaying left bundle branch block morphology, superior axis, and short coupling intervals (320-340 ms), suggestive of short-coupled PVCs (Fig. 1). Transthoracic echocardiography demonstrated preserved left ventricular ejection fraction (63%) and no structural heart disease. Holter monitoring revealed a high PVC burden (2446/24 h), including isolated PVCs, couplets, and 32 episodes of non-sustained PVT, the longest lasting five beats, some triggered by PVCs with coupling intervals <300 ms (Fig. 2). Laboratory testing was unremarkable, myocardial perfusion scintigraphy excluded inducible ischemia, and QT interval was normal (422 ms). Long QT syndrome, catecholaminergic polymorphic VT, structural, and ischemic heart disease were considered unlikely. In the setting of recurrent arrhythmic syncope and short-coupled ventricular ectopy, short-coupled PVT/idiopathic ventricular fibrillation syndrome was considered highly likely. Due to the high risk of recurrent malignant VA, a dual-chamber implantable cardioverter-defibrillator (ICD) was implanted, after shared decision-making. Verapamil 120 mg daily was initiated and later uptitrated after ICD interrogation revealing self-terminating PVT. Despite treatment optimization, she experienced one appropriate ICD shock for ventricular fibrillation-zone arrhythmia (Fig. 3). Reversible causes were excluded, and quinidine 200 mg three times daily was initiated. Genetic testing, including SCN5A, RYR2, and DPP6 variants, was negative.

Discussion

This case highlights short-coupled PVT as a rare but life-threatening cause of syncope in structurally normal hearts. Recognition of short-coupled PVCs is essential for diagnosis. Although verapamil may reduce ventricular ectopy, quinidine may represent an effective treatment option in patients with recurrent VA and/or ICD therapies despite initial medical treatment.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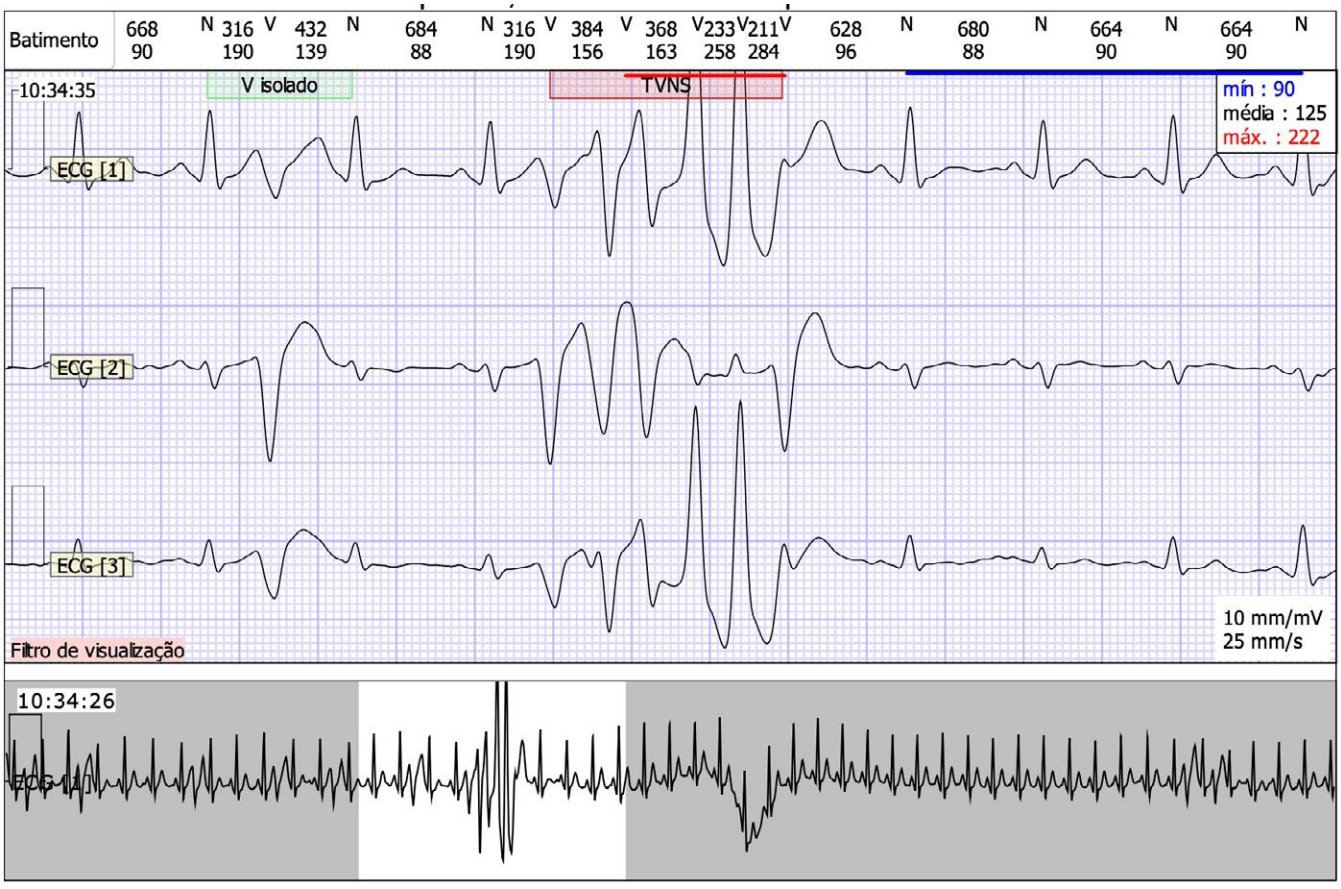
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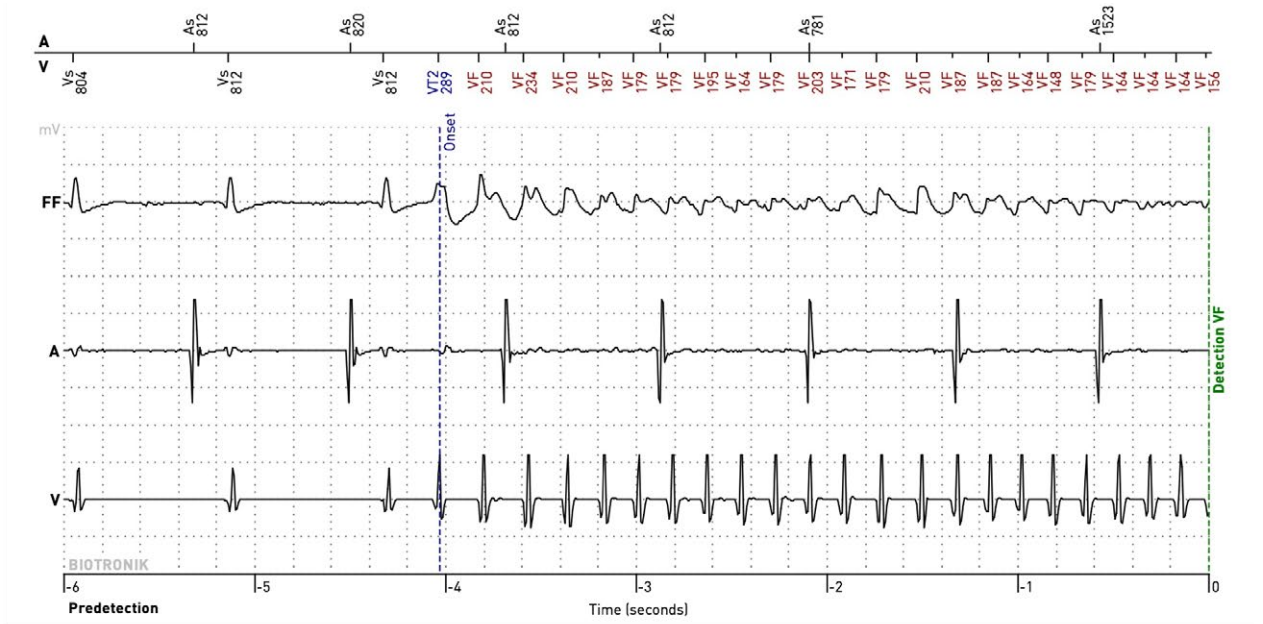
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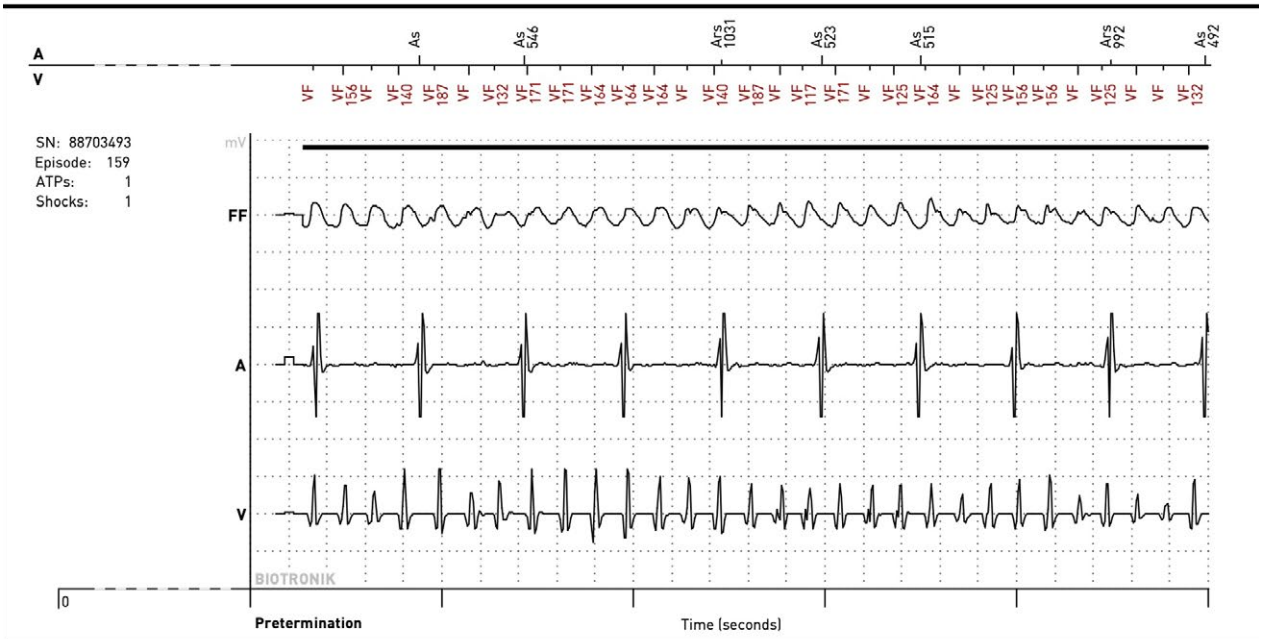
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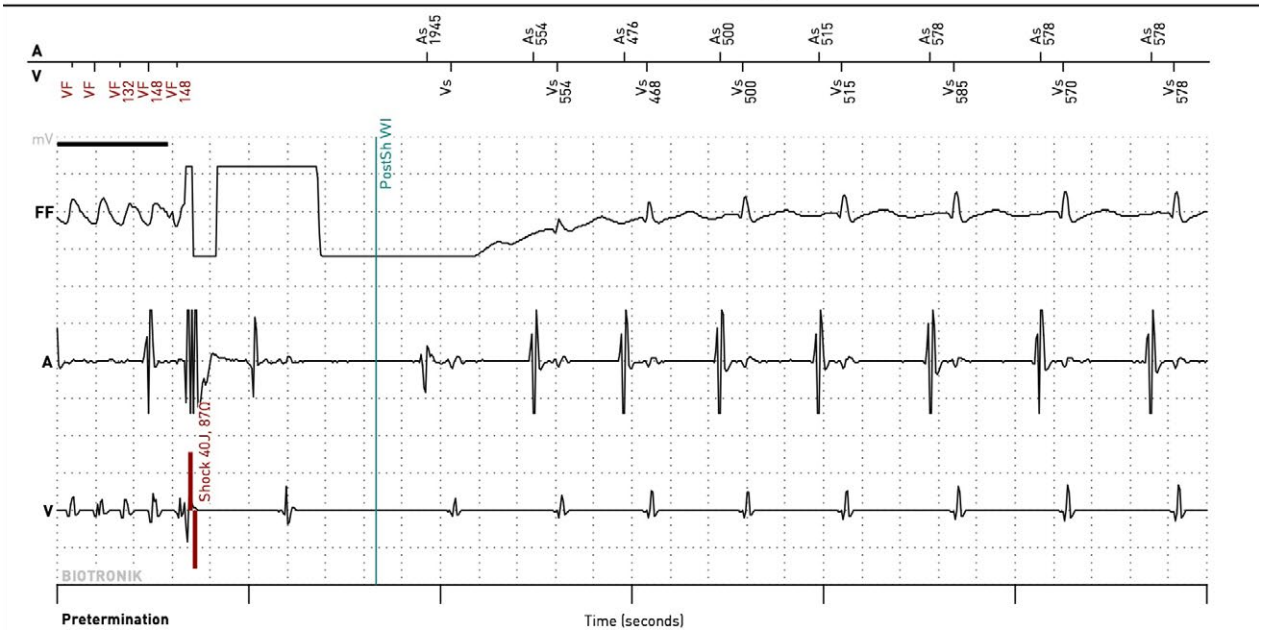
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Supraventricular arrhythmia control with flecainide in arrhythmogenic right ventricular cardiomyopathy with bi-ventricular involvement and mild left ventricular dysfunction

Adriana Vazão; Joana Reis Pereira; André Martins; Mónica Amado; Carolina Esteves; Maksym Baburko; João Carvalho; Hélia Martins; David Durão

UNIDADE LOCAL DE SAÚDE DA REGIÃO DE LEIRIA

Introduction

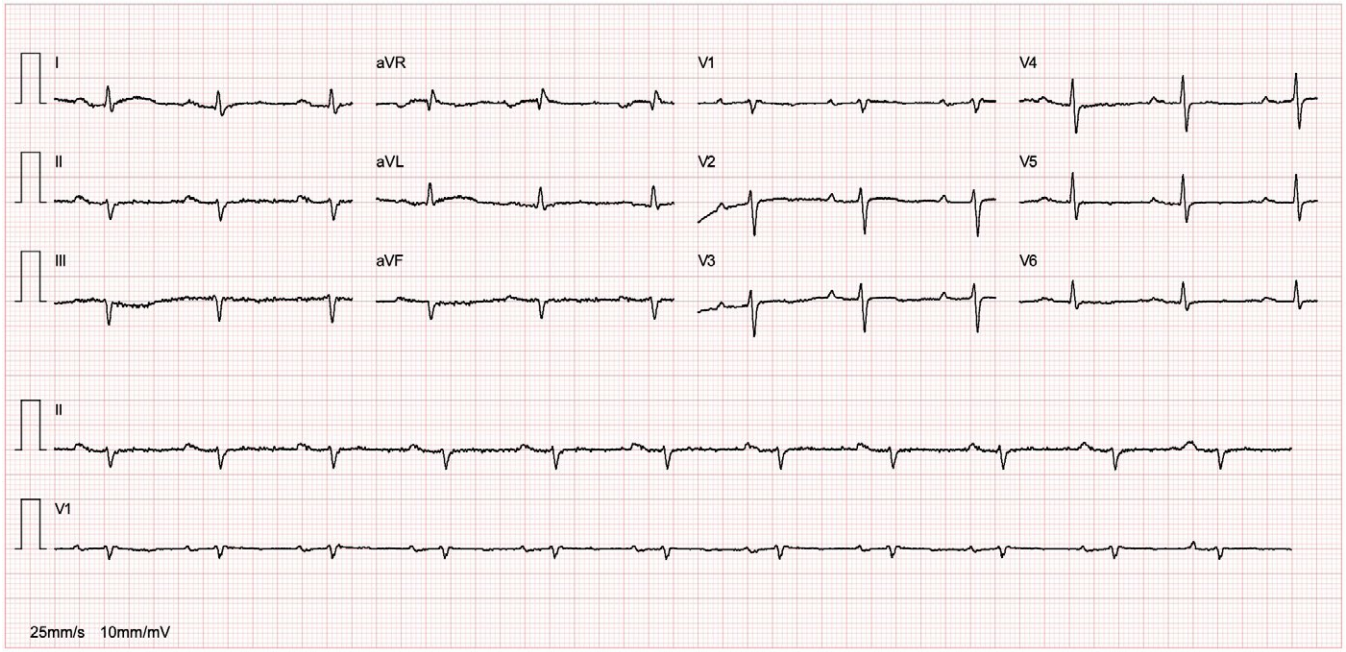
Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an inherited cardiomyopathy associated with supraventricular and ventricular arrhythmias, frequently causing syncope and palpitations. While beta-blockers are first-line therapy and amiodarone is commonly used for arrhythmia control, intolerance may limit long-term treatment. Flecainide may represent a therapeutic option in selected patients.

Case description

A 68-year-old woman with hypertension and hypothyroidism was referred following syncope and a long history of palpitations and dizziness. Baseline electrocardiogram (ECG) showed sinus rhythm with conduction abnormalities and diffuse low-amplitude T waves (Fig. 1). Echocardiography revealed preserved left ventricular ejection fraction with regional wall-motion abnormalities. Holter monitoring demonstrated frequent premature ventricular contractions (2747/24 h) and non-sustained ventricular tachycardia (NSVT). Amiodarone was initiated, with symptomatic improvement. However, repeat Holter monitoring showed sustained supraventricular tachycardia (SVT), frequent polymorphic PVCs, and recurrent NSVT. One month later, ECG during sustained palpitations documented SVT suggestive of atrioventricular nodal reentrant tachycardia (AVNRT). Cardiac magnetic resonance imaging demonstrated biventricular involvement with extensive subepicardial late gadolinium enhancement (Fig. 2), while genetic testing identified a pathogenic desmoplakin mutation, fulfilling revised Padua criteria for definite arrhythmogenic cardiomyopathy. The 2019 ARVC risk score estimated a 5-year ventricular arrhythmia risk of 9.8%, leading to dual-chamber implantable cardioverter-defibrillator implantation for primary prevention. Amiodarone was later discontinued because of intolerance. ICD interrogation subsequently revealed recurrent SVT. Flecainide, introduced by her primary care physician, resulted in symptom resolution, but was discontinued at cardiology follow-up because of concerns regarding its use in structural heart disease. The patient declined catheter ablation (CA) and refused amiodarone re-initiation. Following flecainide withdrawal, recurrent SVT occurred, including episodes misclassified within the ventricular fibrillation detection zone, resulting in anti-tachycardia pacing (Fig. 3) and one inappropriate ICD shock. Given prior benefit, lack of tolerated alternatives, and multidisciplinary discussion, flecainide 50 mg twice daily was reintroduced. Symptoms resolved completely, and ICD interrogation confirmed absence of further supraventricular or ventricular arrhythmias.

Discussion

This case highlights flecainide as a potentially safe and effective option in selected patients with ARVC and complex arrhythmic burden, particularly when amiodarone is poorly tolerated and CA declined.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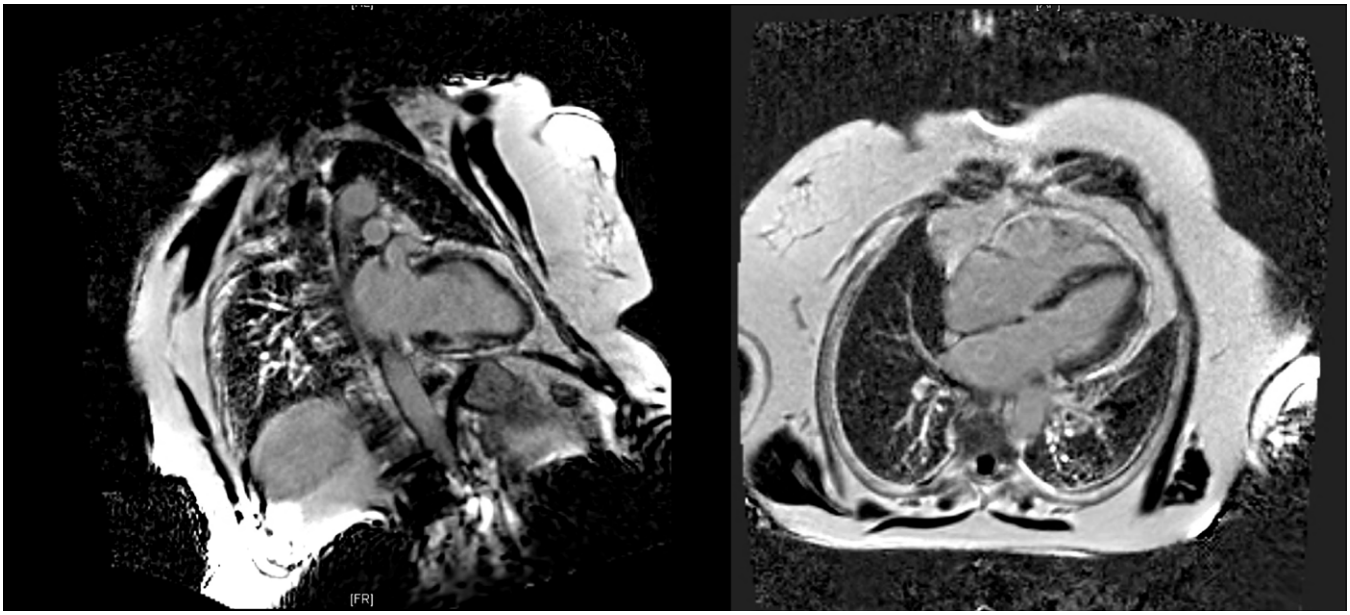
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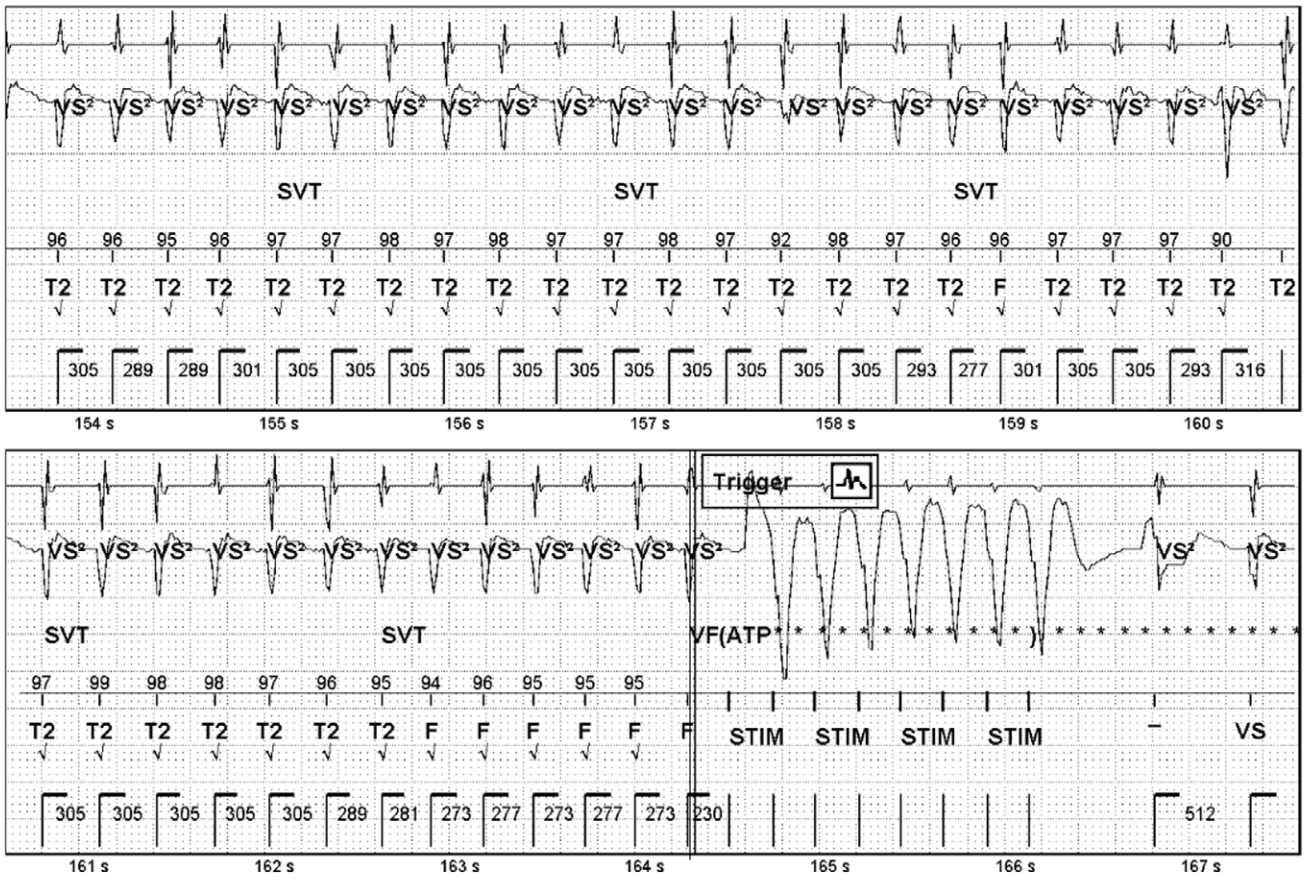

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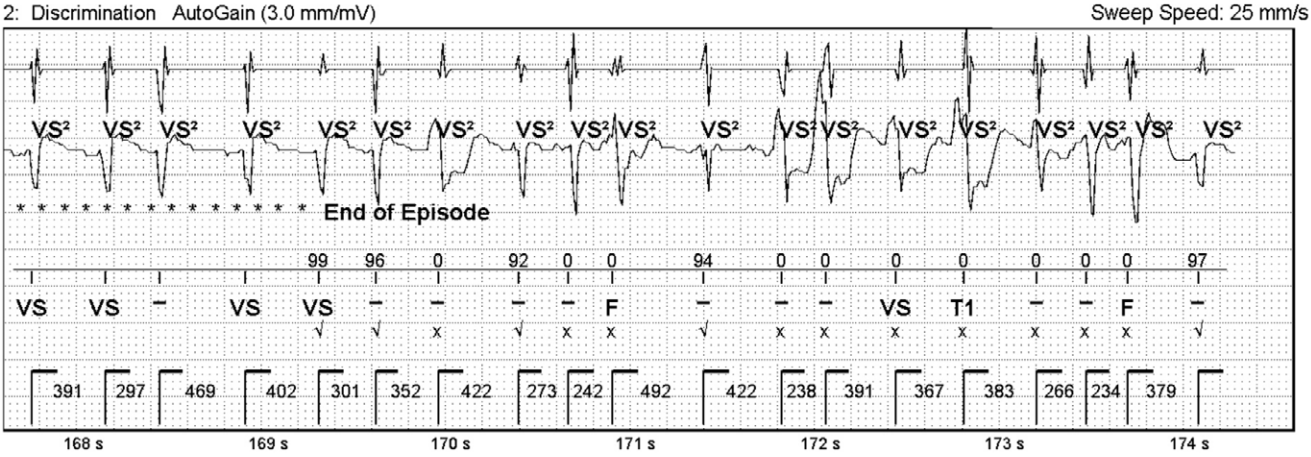


Acute coronary syndrome in the setting of infective endocarditis: an unsuspected culprit

David Campos; Catarina Pohle; Patrícia Bernardes; Marco Tomaz; Ivo Palmeiro; Leonel Pereira; Ana Fátima Esteves; Ricardo Santos; Catarina Sá; Filipe Seixo

UNIDADE LOCAL DE SAÚDE DA ARRÁBIDA

Introduction

Embolic events are potentially life-threatening complications of infective endocarditis (IE), related to migration of cardiac vegetations. Although the brain and spleen are the most frequently affected sites for left-sided IE, other systems may be affected.

Case Report

A 54-year-old male was admitted in the emergency department with disorientation and aphasia for 5 days. Laboratory analysis revealed severe acute kidney injury, requiring haemodialysis, and a left ischemic stroke on initial brain angio-CT.

During haemodialysis the patient developed unstable atrial fibrillation, with peak troponin I (TnI) of 5604 pg/mL. The transthoracic echocardiogram (TTE) revealed preserved left ventricular ejection fraction and extensive calcification of aortic and mitral valves, without significant functional changes.

On repeat brain angio-CT there was evidence of haemorragic transformation, which delayed the coronary angiography for two weeks; it revealed a significant mid-right coronary artery (RCA) stenosis which was treated with drug-eluting stent (DES) implantation.

At day 29, blood cultures were positive for methicillin-resistant Staphylococcus aureus (MRSA), and vancomycin was started. At day 33, there was recurrent angina with peak TnI of 13000 pg/mL. A transoesophageal echocardiogram (TOE) revealed a 11mm vegetation on the posterior leaflet of the mitral valve.

At day 45, the patient complained of recurrent chest pain and a de novo large pericardial effusion was detected, progressing to cardiac tamponade. Pericardiocentesis drained 940mL of hematic fluid, with cultures positive for MRSA. The case was discussed in Heart Team but the patient was refused for surgery.

Due to recurrent episodes of angina and re-elevation of TnI, coronary angiography was repeated, showing contrast extravasation and contained perforation of the mid-RCA, which was successfully treated with covered stents implantation.

The patient remained stable and asymptomatic and was discharged under antibiotic treatment at day 91.

Discussion

This case demonstrates a possible coronary rupture due to septic embolization. Although IE of the mitral valve was only confirmed at day 35, prior TTE already identified extensive valve changes that could be related to subclinical IE. Coronary rupture seems to explain the haematic pericardial fluid, and due to the timeframe between coronary angiography and pericardial effusion (12 days), this complication might be due to septic embolization with subsequent development of mycotic aneurysms.

This case highlights the importance of recognizing blood stream infections and IE as a source of coronary complications, which should lead to frequent echocardiographic monitoring and extended clinical vigilance.

Calcific Constrictive Pericarditis with Marked Ventricular Deformation Mimicking Hepatic Decompensation

Joana Pereira; Adriana Vazão; Mónica Amado; André Martins; Carolina Esteves; Mariana Saraiva; Davide Severino; David Durão

UNIDADE LOCAL DE SAÚDE DA REGIÃO DE LEIRIA

Introduction

Constrictive pericarditis (CP) is an uncommon but potentially curable cause of right-sided heart failure. Diagnosis may be challenging in patients with chronic liver disease, where ascites, pleural effusion, and peripheral edema are often attributed to hepatic decompensation. We report a case of advanced calcific CP presenting as presumed hepatic decompensation, with remarkable biventricular deformation demonstrated by multimodality imaging.

Case Description

A 52-year-old man with hypertension and chronic liver disease related to previous hepatitis C and alcohol exposure presented with progressive edema, ascites, dyspnea, orthopnea, and functional decline over 3 months. On admission, he exhibited severe anasarca, massive pleural effusion, ascites, respiratory failure, and oliguric acute kidney injury (creatinine 5.96 mg/dL). The initial diagnosis was hepatic decompensation.

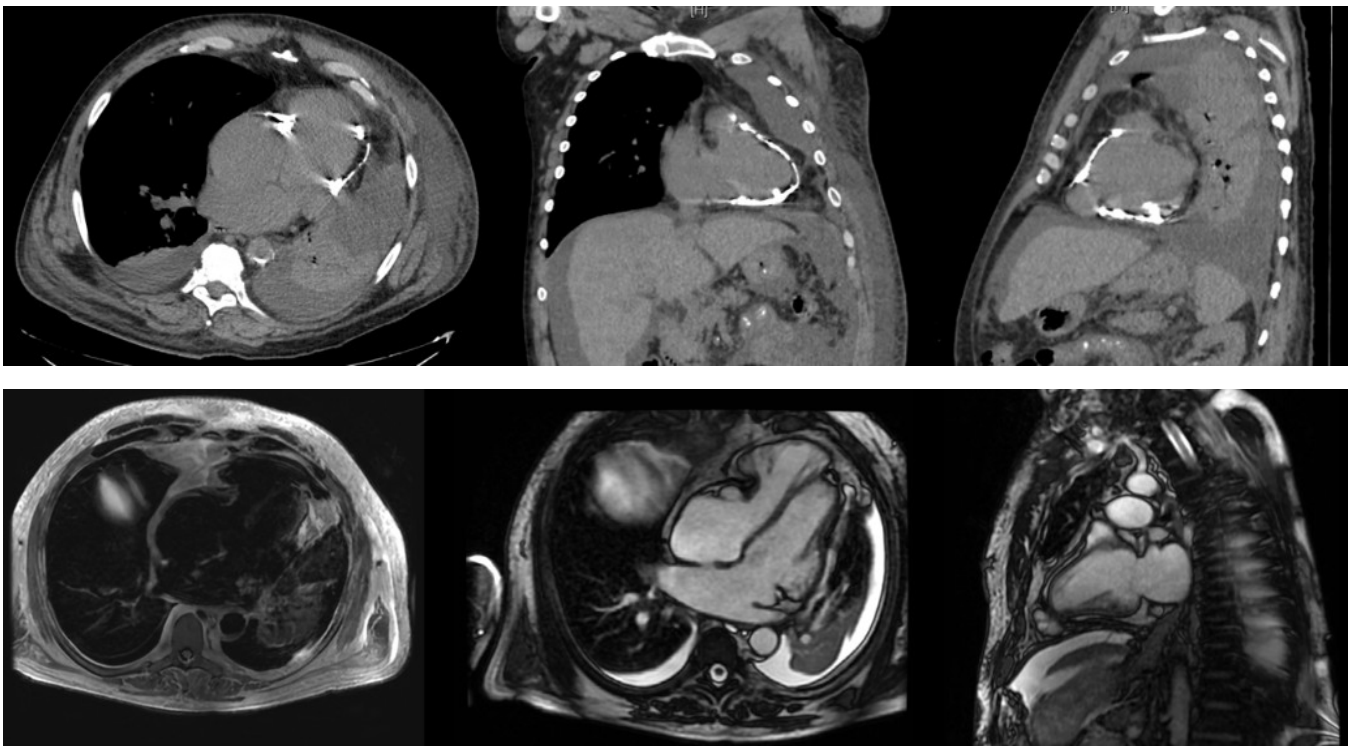
Preserved liver synthetic function prompted further investigation. Electrocardiography showed low QRS voltage. Computed tomography revealed extensive circumferential pericardial calcification, pleural effusions, ascites, and an unusual ventricular configuration. Echocardiography demonstrated paradoxical septal motion, biatrial enlargement, dilated inferior vena cava, and fibrocalcific pericardial abnormalities.

Cardiac magnetic resonance was decisive, showing marked pericardial thickening with severe deformation of both ventricles. The left ventricle displayed a conical configuration with restricted diastolic expansion, while the right ventricle showed basal constriction and aneurysmal remodeling of the mid and apical segments. Septal bounce, dilated caval and hepatic veins, and preserved systolic function supported constrictive physiology. Right and left heart catheterization confirmed constrictive pericarditis with post-capillary pulmonary hypertension.

After aggressive decongestive therapy, renal function normalized and respiratory failure resolved. The patient subsequently underwent pericardiectomy. Histopathology demonstrated fibrous thickening with extensive dystrophic calcification, confirming chronic calcific constrictive pericarditis.

Discussion

This case illustrates how constrictive pericarditis may masquerade as hepatic decompensation in patients with underlying liver disease. Multimodality imaging was essential for diagnosis and revealed not only classical constrictive physiology but also striking biventricular deformation caused by chronic calcific pericardial encasement. The unusual ventricular geometry, particularly the aneurysmal appearance of the right ventricle, highlights the capacity of advanced calcific CP to remodel cardiac structure beyond its hemodynamic effects. Early recognition is crucial, as definitive surgical treatment may be curative.



Acute Pericarditis Unmasking High-Risk Obstructive Hypertrophic Cardiomyopathy in a Young Adult

David Campos; Catarina Pohle; Patrícia Bernardes; Marco Tomaz; Ivo Palmeiro; Leonel Pereira; Jéni Quintal; Rui Coelho; Dinis Mesquita; Filipe Seixo

UNIDADE LOCAL DE SAÚDE DA ARRÁBIDA

Introduction

Recent cardiac magnetic resonance (CMR) studies suggest that hypertrophic cardiomyopathy (HCM) may be more prevalent than previously recognized. Early diagnosis and risk stratification are crucial to prevent adverse outcomes, particularly sudden cardiac death (SCD).

Case Presentation

A 24-year-old male with no previous medical history presented to the emergency room with pleuritic chest pain relieved by leaning forward. Laboratory testing showed a peak high-sensitivity troponin of 54 pg/mL and C-reactive protein <0.1 mg/dL.

Bedside transthoracic echocardiography (TTE) revealed asymmetric left ventricular hypertrophy, predominantly involving the interventricular septum (18-19 mm) and apex, with an "ace-of-spades" shape. Left ventricular outflow tract obstruction (LVOTO) was present, with peak gradients of 32 mmHg at rest and 50 mmHg during Valsalva manoeuvre. Ejection fraction was preserved.

An inflammatory myopericardial syndrome versus symptomatic HCM was initially considered. The patient was admitted and started on beta-blocker and anti-inflammatory therapy, with symptom resolution. During hospitalization, a single episode of non-sustained ventricular tachycardia was documented.

Repeat TTE on day 7 demonstrated improvement of LVOTO gradients (22 mmHg at rest and 34 mmHg during Valsalva manoeuvre).

CMR performed on day 10 showed severe asymmetric hypertrophy, predominantly affecting the basal anterior wall, with a maximal wall thickness of 31 mm and preserved ejection fraction (74%). T2 mapping demonstrated no myocardial oedema. Late gadolinium enhancement revealed nodular and mid-wall fibrosis within the basal anterior segment and at the right ventricular insertion points. A mild pericardial effusion (5 mm) was also identified.

Exercise stress echocardiography showed excellent functional capacity without symptoms or haemodynamic instability. However, dynamic LVOTO increased markedly, to a peak gradient of 127 mmHg.

Acute pericarditis with myocardial injury was diagnosed, while active myocarditis was considered unlikely given the absence of myocardial oedema on CMR. Concomitantly, the patient was diagnosed with asymptomatic obstructive HCM.

Given an HCM Risk-SCD score of 14.6% and documented non-sustained ventricular tachycardia, he was referred to a specialized inherited cardiomyopathy clinic and scheduled for implantable cardioverter-defibrillator implantation.

Discussion

This case highlights the importance of multimodality imaging in differentiating inflammatory cardiac syndromes from structural cardiomyopathies. Although the clinical presentation was suggestive of acute pericarditis, comprehensive evaluation revealed previously undiagnosed obstructive HCM with high-risk features for SCD. Early recognition and appropriate risk stratification were essential to guide potentially life-saving therapy.

16TH CHALLENGES IN CARDIOLOGY

IMAGES

A Fragile Aorta: Endovascular Treatment of a Mycotic Aneurysm

Andreia Lopes Sousa; Filipe Vilela; Bárbara Rocha; João Faria; Carla Oliveira Ferreira; Mónica Dias; Sofia Fernandes; Rui Files Flores

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

Mycotic aortic aneurysm is a rare but potentially fatal vascular infection that leads to dilation or pseudoaneurysm formation of the aorta (most commonly at the infrarrenal abdominal aorta). It is associated with a high risk of rupture and death, particularly in patients with pre-existing cardiovascular disease. Diagnosis is often challenging because clinical features are non-specific, so a high index of suspicion is required, especially in patients with bacteraemia. Early recognition and prompt treatment are essential, as delays in management are linked to poor outcomes.

Clinical case

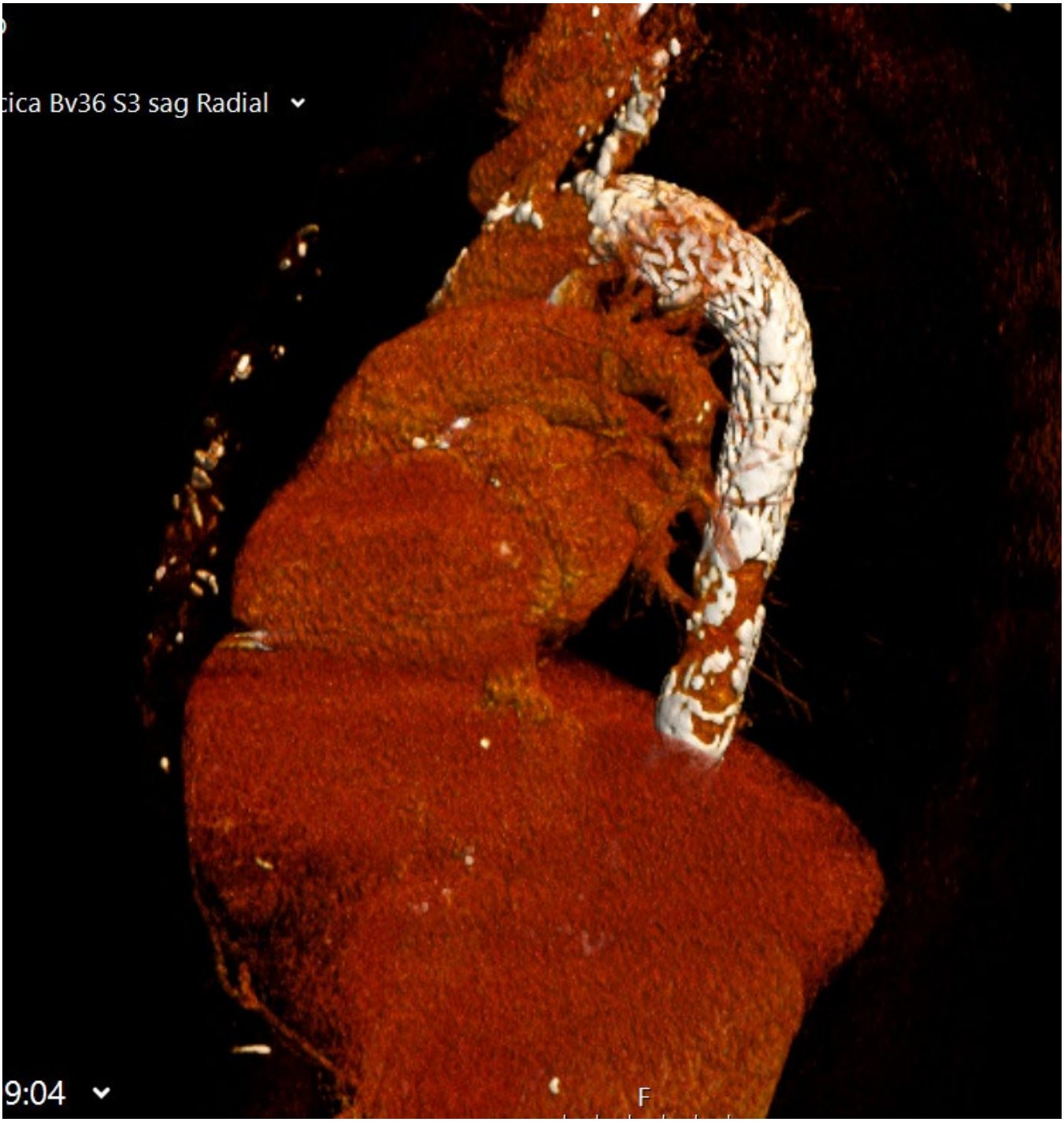
A 70-year-old man with a history of coronary and peripheral artery disease presented to the emergency department with a 4-day history of fever and chest pain. Laboratory tests showed markedly elevated C-reactive protein and leukocytosis. Two sets of blood cultures were positive for methicillin-sensitive *Staphylococcus aureus*. The patient was admitted to the Infectious Diseases ward for further workup.

Thoracic CT angiography revealed irregular, calcified and ulcerated plaques in the distal portion of the aortic arch consistent with infective aortitis with possible mycotic aneurysm. Given the severe calcification affecting all the major arterial vessels, cardiothoracic surgery considered open repair with vascular anastomosis unfeasible, so a conservative and endovascular approach was therefore selected. The patient underwent thoracic endovascular aortic repair two days after the diagnosis, without complications.

He completed a course of 7 weeks of antibiotic therapy, with complete resolution of symptoms. Supressive antibiotherapy was considered. Follow-up CT showed complete resolution of the infectious process with no prosthetic involvement detected. A positron emission tomography is scheduled.

Discussion

Thoracic mycotic aneurysm remains a highly lethal condition despite treatment. In patients with a contraindication to open surgical repair, thoracic endovascular aortic repair is becoming recognized as a feasible and safe alternative yet requiring a close follow-up. Atypical locations, mainly in patients with severe atherosclerotic disease, must not be overregarded as potentially associated with infectious nature.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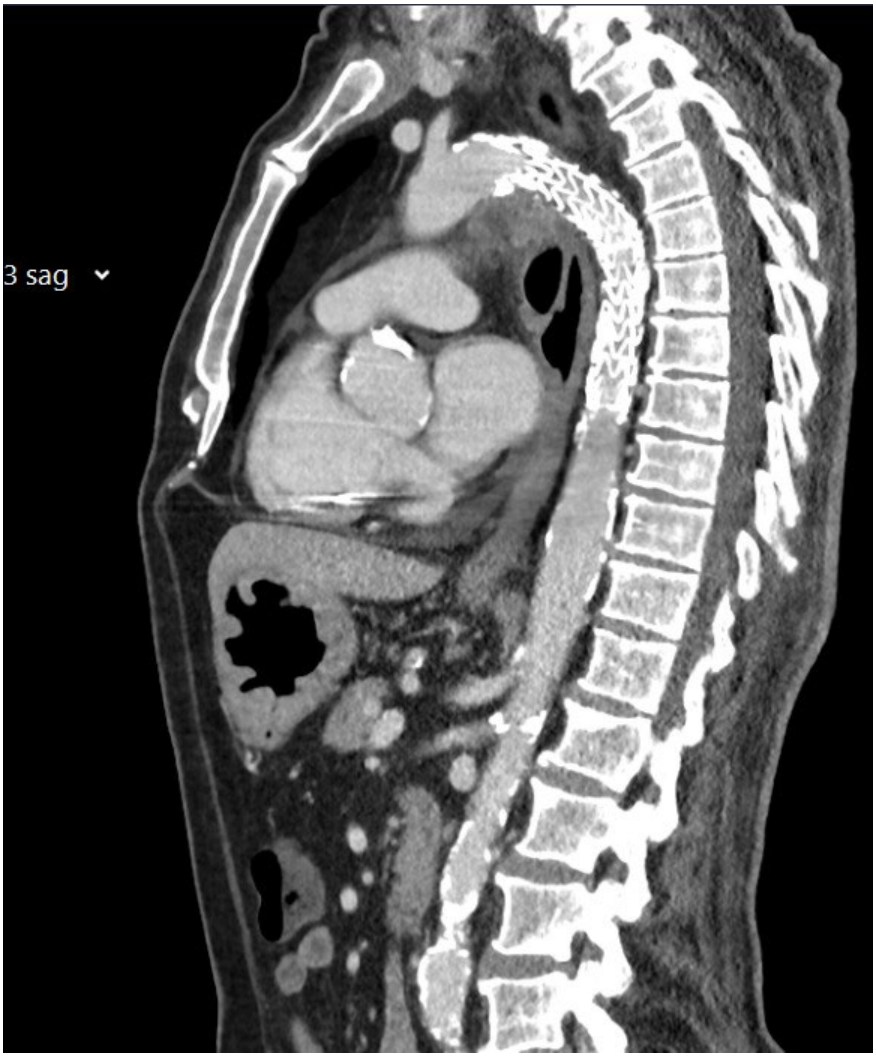
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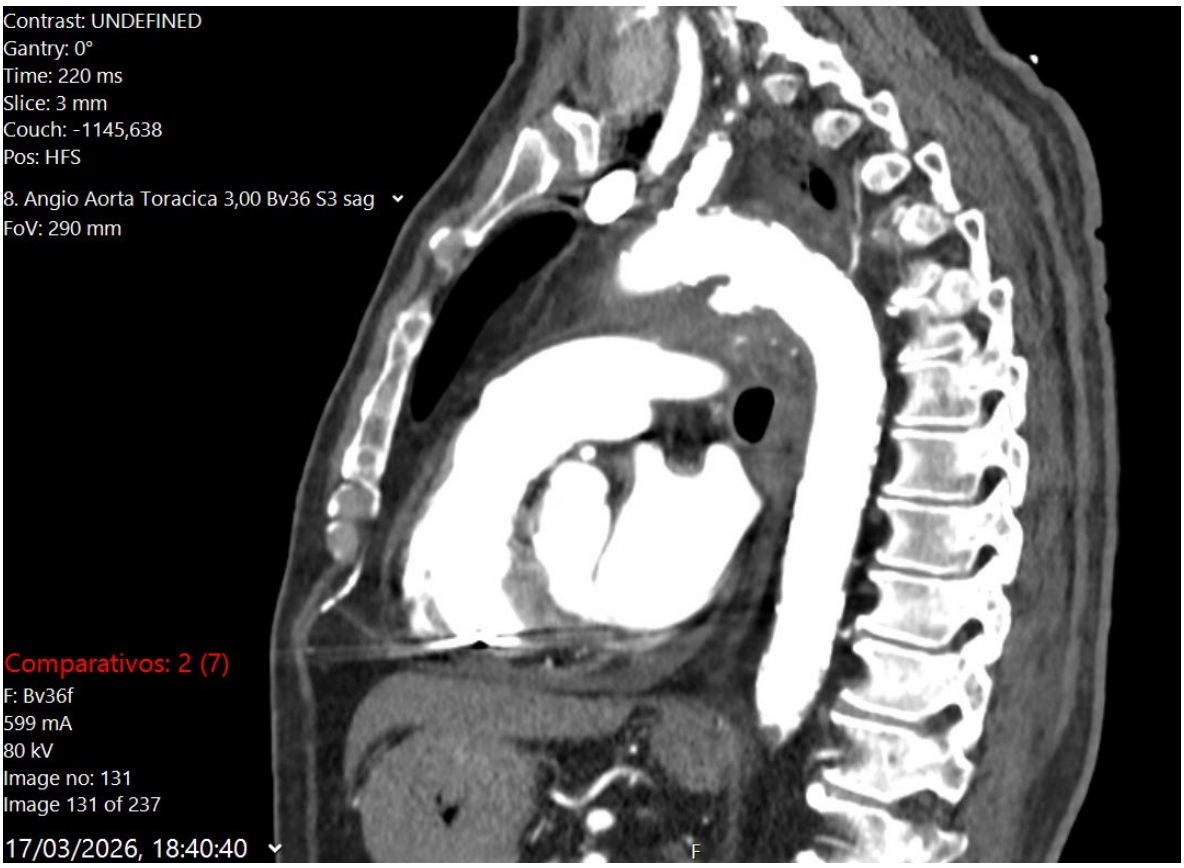
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Arrhythmogenic Mitral Valve: When Structure Drives Sudden Death

Bárbara Antunes Rocha; João Faria; Andreia Sousa; Carla Ferreira; Filipe Vilela; Mónica Dias; Sofia Fernandes; Vítor Hugo Pereira; Catarina Vieira; Rui Files Flores; Carlos Galvão Braga

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

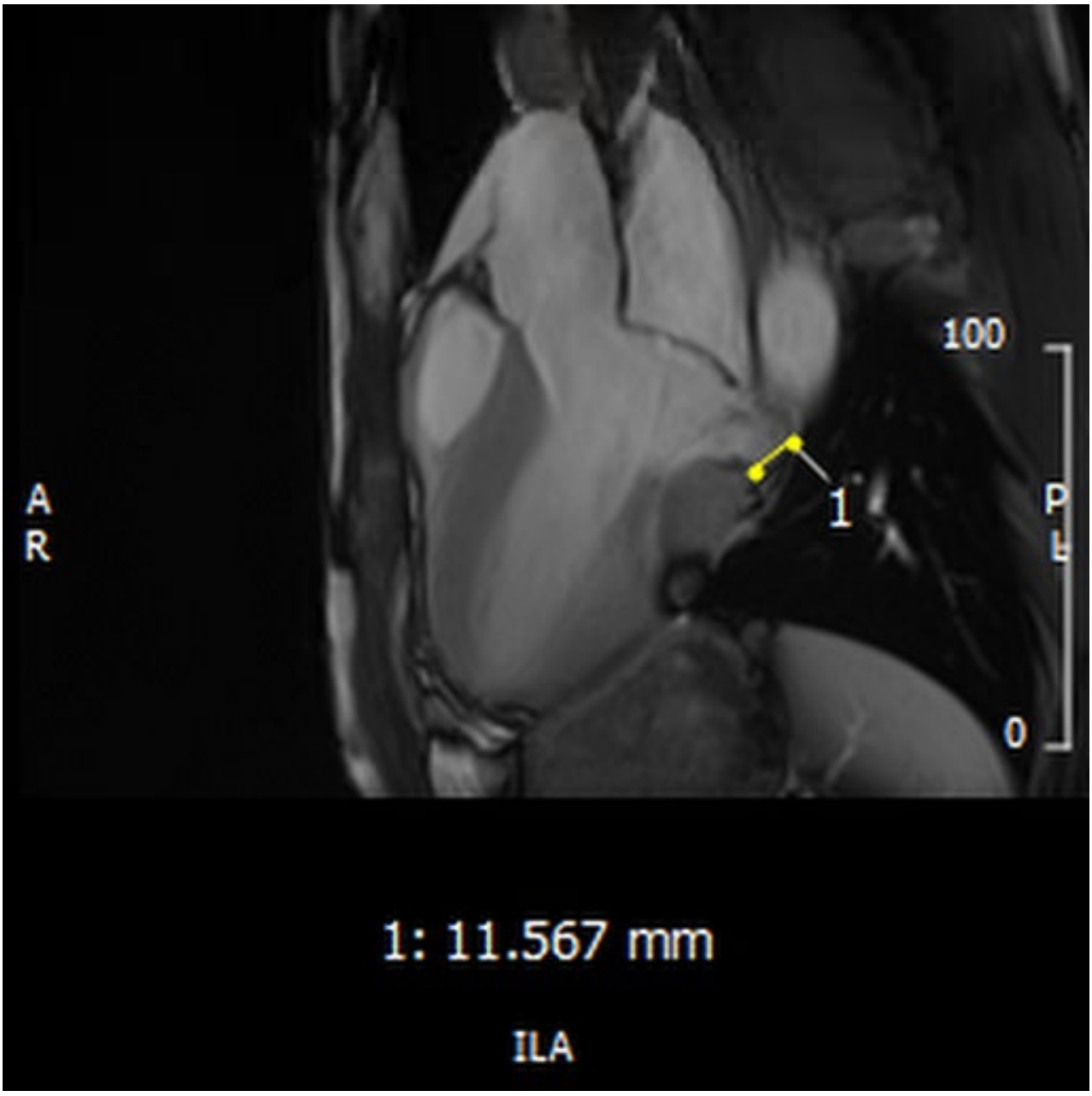
Mitral annular disjunction (MAD) is a structural abnormality defined by atrial displacement of the mitral valve leaflet hinge point relative to the left ventricular myocardium, frequently associated with myxomatous mitral valve disease and mitral valve prolapse (MVP). Once considered benign, MAD is now recognised as a substrate for malignant ventricular arrhythmias, including ventricular fibrillation and sudden cardiac death, even without significant mitral regurgitation or ventricular dysfunction. Proposed mechanisms include abnormal mechanical stress at the mitral annulus, basal inferolateral fibrosis, and systolic curling of the basal inferolateral wall. The Pickelhaube sign, a tissue Doppler-derived marker, reflects abnormal annular motion beyond simple hypermobility. Cardiac magnetic resonance (CMR) enables anatomical and tissue characterisation, identifying high-risk phenotypes.

Case Report

A 26-year-old man with known MAD and myxomatous MVP, under irregular beta-blocker therapy, presented after out-of-hospital cardiac arrest due to ventricular fibrillation. On admission, he was haemodynamically stable but comatose, requiring intubation. Post-resuscitation ECG showed no features suggestive of primary electrical disease. Transthoracic echocardiography revealed a non-dilated, non-hypertrophied left ventricle with preserved biventricular systolic function, multiscallop bileaflet myxomatous MVP, mild mitral regurgitation, MAD with 5 mm excursion, and the Pickelhaube sign. Coronary angiography excluded obstructive disease. CMR confirmed preserved ventricular function, bileaflet MVP with MAD, basal inferolateral systolic curling, and non-ischaemic mid-wall fibrosis involving the inferior and inferolateral walls, consistent with an arrhythmogenic mitral valve phenotype. A subcutaneous ICD was implanted for secondary prevention, and beta-blocker therapy was optimised with bisoprolol. The patient remained free from recurrent arrhythmic events during follow-up.

Conclusion

This case illustrates the risk of sudden cardiac death in patients with MAD and MVP despite preserved ventricular function. Risk stratification is inherently multiparametric, integrating clinical variables, ECG and Holter findings, and comprehensive echocardiographic assessment including tissue Doppler imaging and myocardial strain, alongside CMR and PET-CMR data. The combination of bileaflet prolapse, annular disjunction, Pickelhaube sign, and CMR-detected fibrosis defines a high-risk arrhythmogenic phenotype. This case underscores the importance of systematic risk stratification to identify patients at highest arrhythmic risk, enabling closer surveillance and timely intervention.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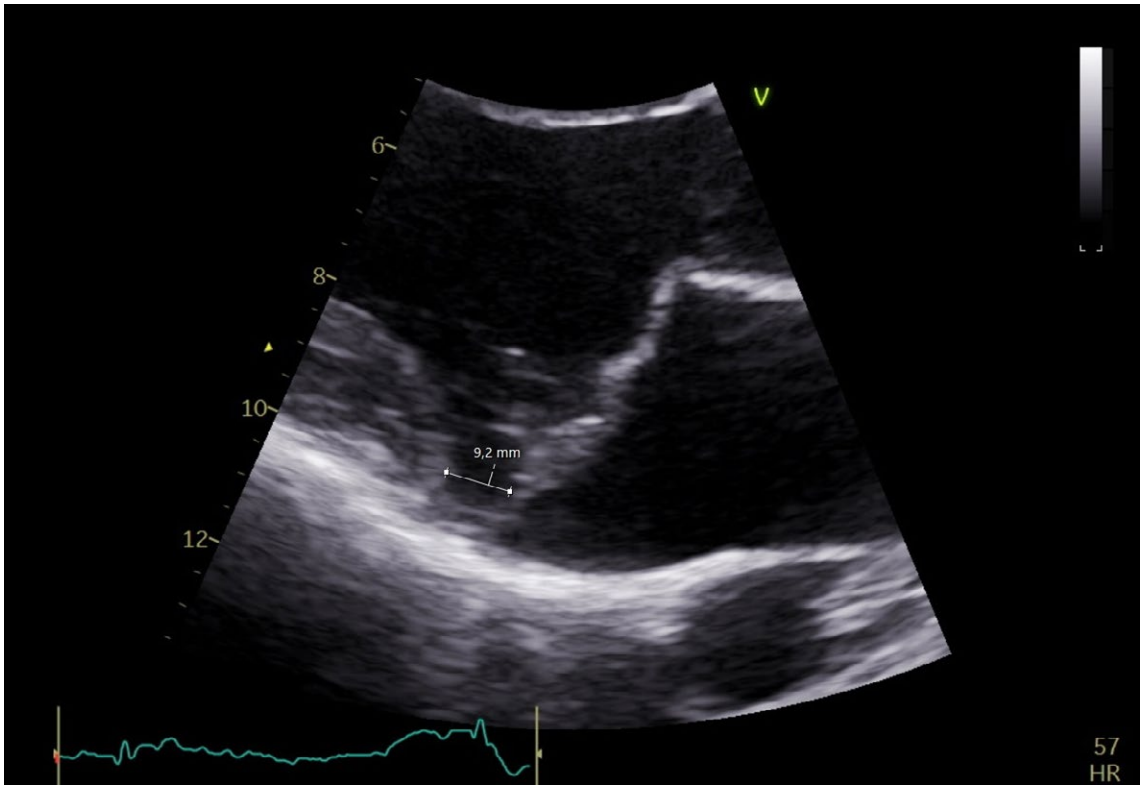
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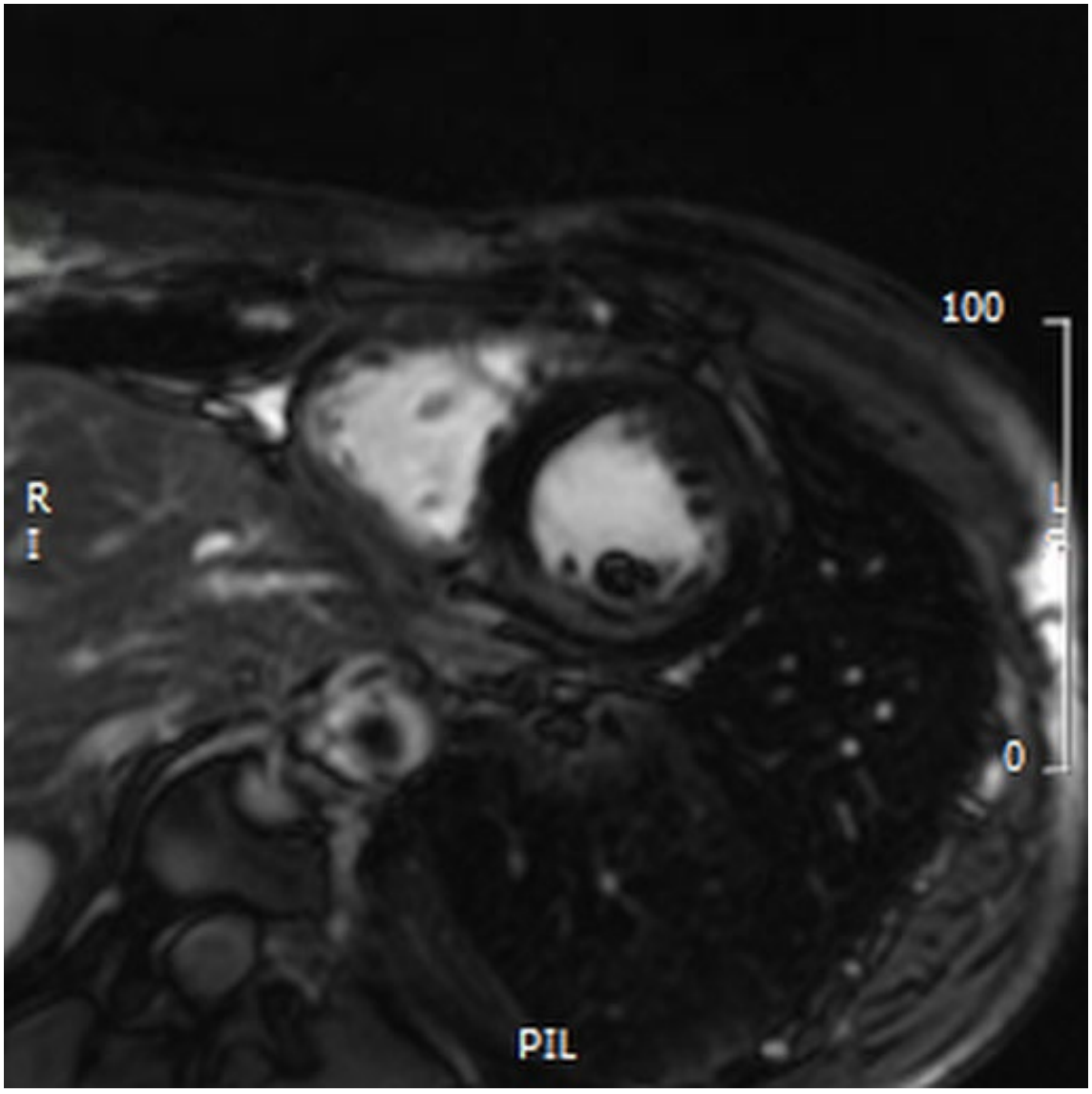
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Platypnea-Orthodeoxia or Something Else? The Uncertainty Behind a Therapeutic Decision

Bárbara Antunes Rocha; João Faria; Andreia Sousa; Carla Ferreira; Filipe Vilela; Mónica Dias; Sofia Fernandes; Carlos Galvão Braga

UNIDADE LOCAL DE SAÚDE DE BRAGA

Introduction

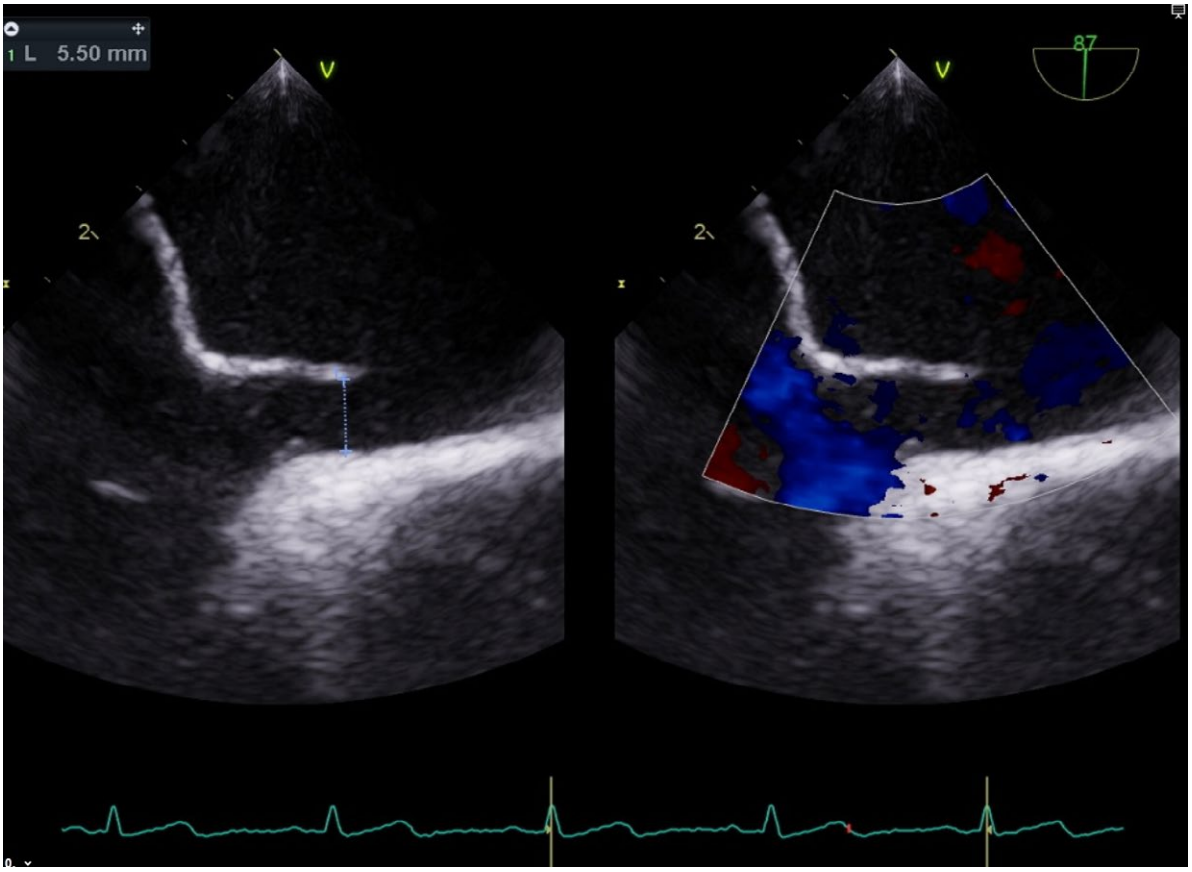
Platypnea-orthodeoxia syndrome (POS) is a rare condition characterized by positional dyspnea and arterial desaturation in the upright position, improving when supine. It is most commonly associated with intracardiac shunts, particularly patent foramen ovale (PFO), often triggered by concomitant pulmonary or structural cardiac abnormalities. Early diagnosis is essential, as closure of the interatrial defect may result in significant clinical improvement.

Case report

A 77-year-old woman with arterial hypertension, atrial fibrillation, and chronic eosinophilic pneumonia presented with resting dyspnea and marked asthenia. She was hemodynamically stable but tachypneic, requiring supplemental oxygen. Pulmonary examination revealed bibasilar crackles and bilateral ankle edema. Arterial blood gas analysis showed hypoxemic respiratory failure. Laboratory tests demonstrated mild anemia, hyponatremia, elevated NT-proBNP, preserved renal function, and no inflammatory markers. Thoracic CT angiography revealed pneumomediastinum, scattered ground-glass opacities, and bilateral subpleural reticulation suggestive of fibrotic lung disease. The patient was admitted for stabilization and optimization of diuretic therapy. Bronchoscopy showed no significant abnormalities. Despite improvement of pulmonary congestion and radiological resolution of the pneumomediastinum, hypoxemia persisted, with recurrent desaturation in the upright position that improved when supine. POS was suspected. Transthoracic echocardiography demonstrated preserved biventricular systolic function, a redundant interatrial septum, and extrinsic compression of the right-sided cardiac chambers, particularly the right atrium. A small right-to-left bubble passage was observed only in the sitting position, but findings were considered inconclusive and no intervention was performed. The patient remained hospitalized with persistent symptoms. Following worsening of the pneumomediastinum, repeat echocardiography showed early passage of a large number of bubbles from the right to the left chambers, confirming a significant intracardiac shunt. After Heart Team discussion, percutaneous PFO closure was successfully performed, resulting in marked clinical improvement and discontinuation of oxygen therapy.

Conclusion

This case highlights the diagnostic challenge of POS in the presence of concomitant pulmonary disease and pneumomediastinum. The evolving clinical course delayed recognition of the intracardiac shunt, emphasizing the value of repeated imaging and multidisciplinary assessment. The findings suggest that pneumomediastinum contributed to a dynamic increase in right-sided chamber compression, promoting right-to-left shunting through a PFO. Percutaneous closure resulted in substantial symptomatic and functional recovery.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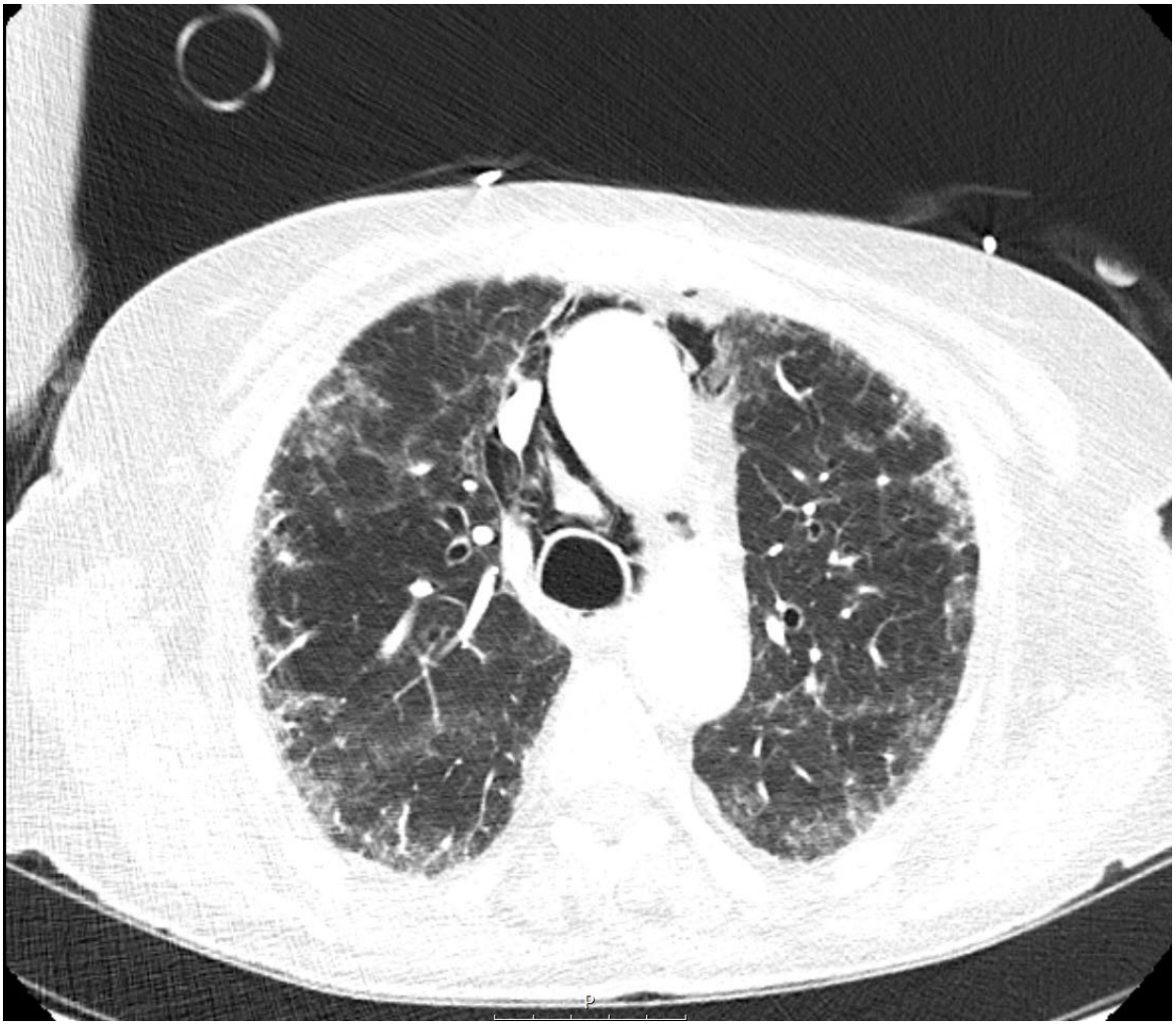


When Imaging Tells the Whole Story: A Giant Left Atrial Myxoma

Joana Pereira; Mónica Amado; Adriana Vazão; André Martins; Carolina Esteves; Luís Santos; Davide Severino; David Durão

UNIDADE LOCAL DE SAÚDE DA REGIÃO DE LEIRIA

Introduction

Cardiac myxoma is the most common primary cardiac tumour in adults and typically arises in the left atrium. Although symptoms may result from embolization or intracardiac obstruction, some patients remain asymptomatic despite large tumour burden. We present the case of a giant left atrial myxoma incidentally detected during routine cardiac imaging.

Case Report

A 62-year-old woman with hypertension, type 2 diabetes mellitus, obesity, and hypothyroidism was referred following the incidental discovery of a left atrial mass on routine transthoracic echocardiography performed by her primary care physician. She denied dyspnoea, chest pain, syncope, palpitations, or exercise intolerance.

Transthoracic echocardiography demonstrated severe left atrial enlargement and a mobile pedunculated mass measuring 55 × 27 mm attached to the interatrial septum, suggestive of myxoma.

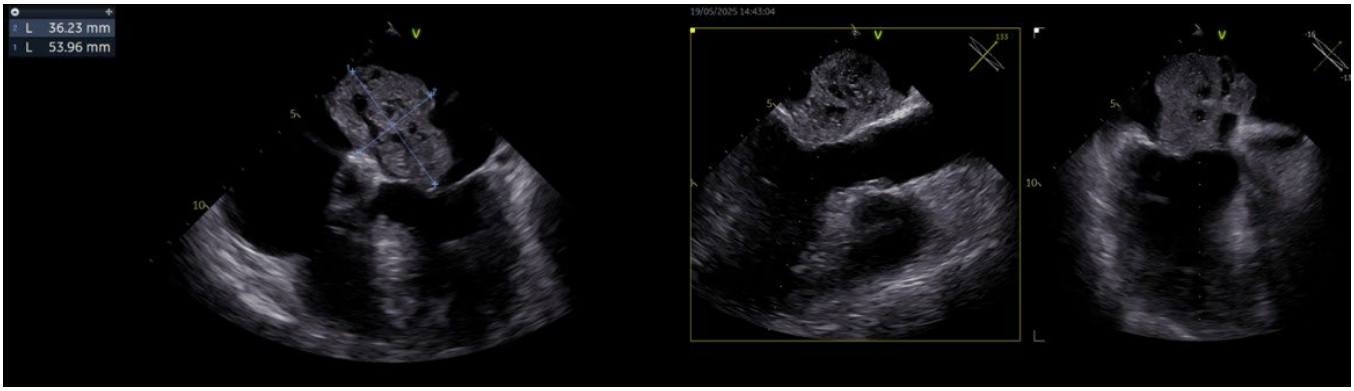
Transoesophageal echocardiography revealed a heterogeneous echogenic mass measuring approximately 54 × 36 mm, attached to the anterosuperior interatrial septum and protruding towards the mitral valve during diastole. The lesion produced functional transmitral obstruction with a peak velocity of 1.8 m/s and a mean gradient of 6 mmHg.

Cardiac CT demonstrated a multilobulated heterogeneous mass measuring 55 × 48 × 40 mm with low attenuation, marked internal vascularization, and delayed contrast retention. The lesion appeared attached to the interatrial septum and anteroseptal wall of the left atrium, exhibiting partial prolapse through the mitral valve. A prominent internal vascular network supplied by collateral branches from both coronary arteries produced a striking "vascular web" appearance. Coronary angiography excluded obstructive coronary artery disease.

The patient underwent successful surgical excision without complications. Histopathological examination confirmed the diagnosis of atrial myxoma. Follow-up echocardiography showed complete resection and no evidence of recurrence.

Discussion and Conclusion

This case illustrates the unusual presentation of a giant left atrial myxoma causing functional mitral inflow obstruction in an entirely asymptomatic patient. Multimodality imaging was crucial for diagnosis and surgical planning. Echocardiography accurately defined tumour mobility and haemodynamic impact, while cardiac CT provided detailed anatomical and vascular characterization, particularly the extensive neovascularization arising from bilateral coronary collaterals. The case highlights the value of routine cardiac imaging in detecting clinically silent cardiac tumours and demonstrates the complementary role of echocardiography and cardiac CT in the assessment of atrial myxomas.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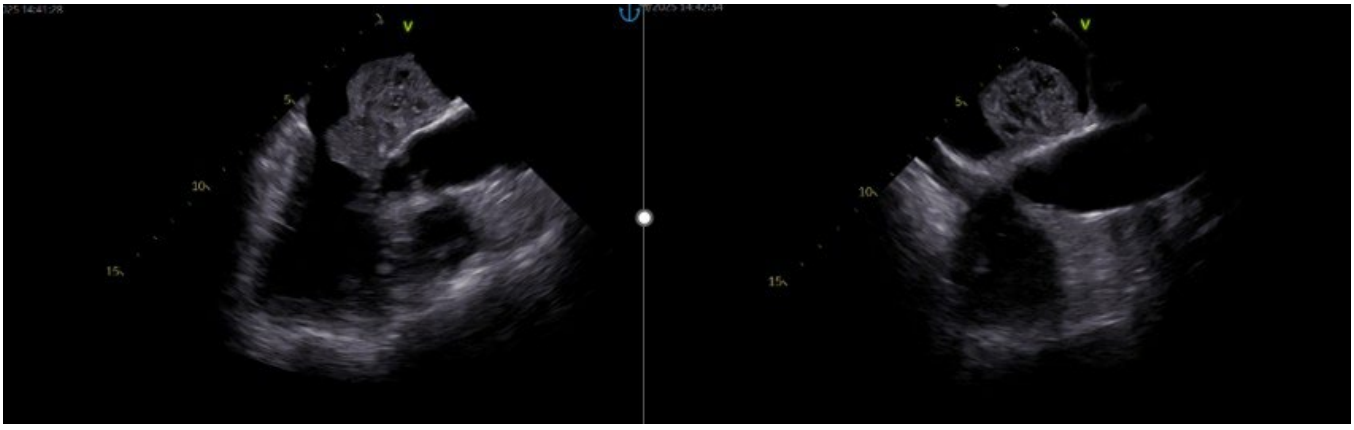
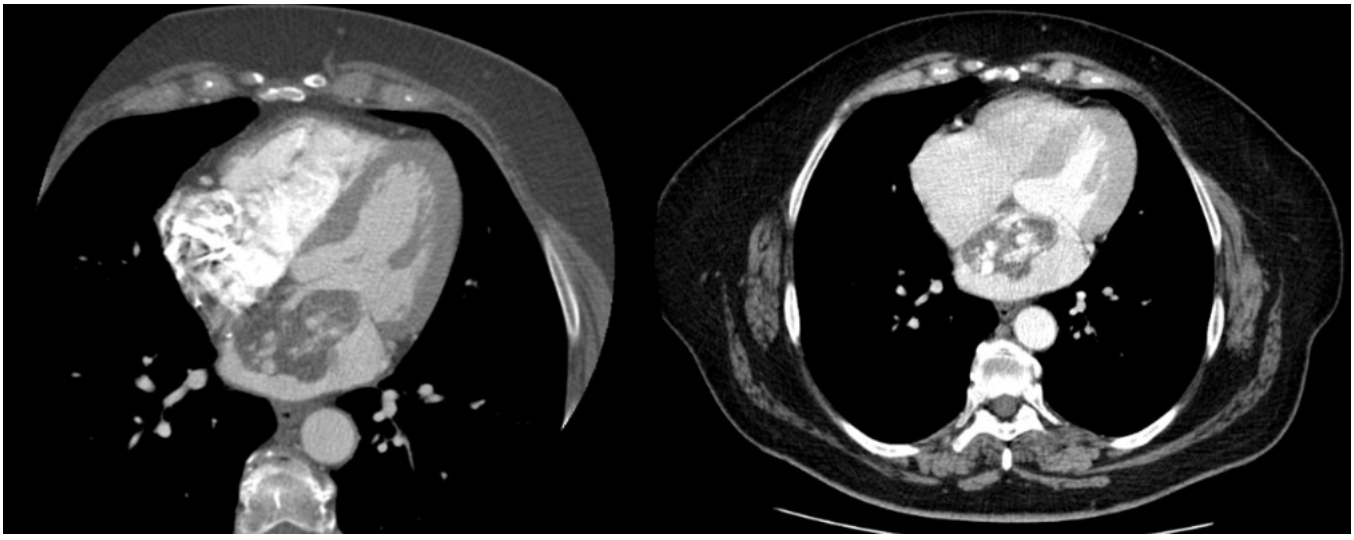
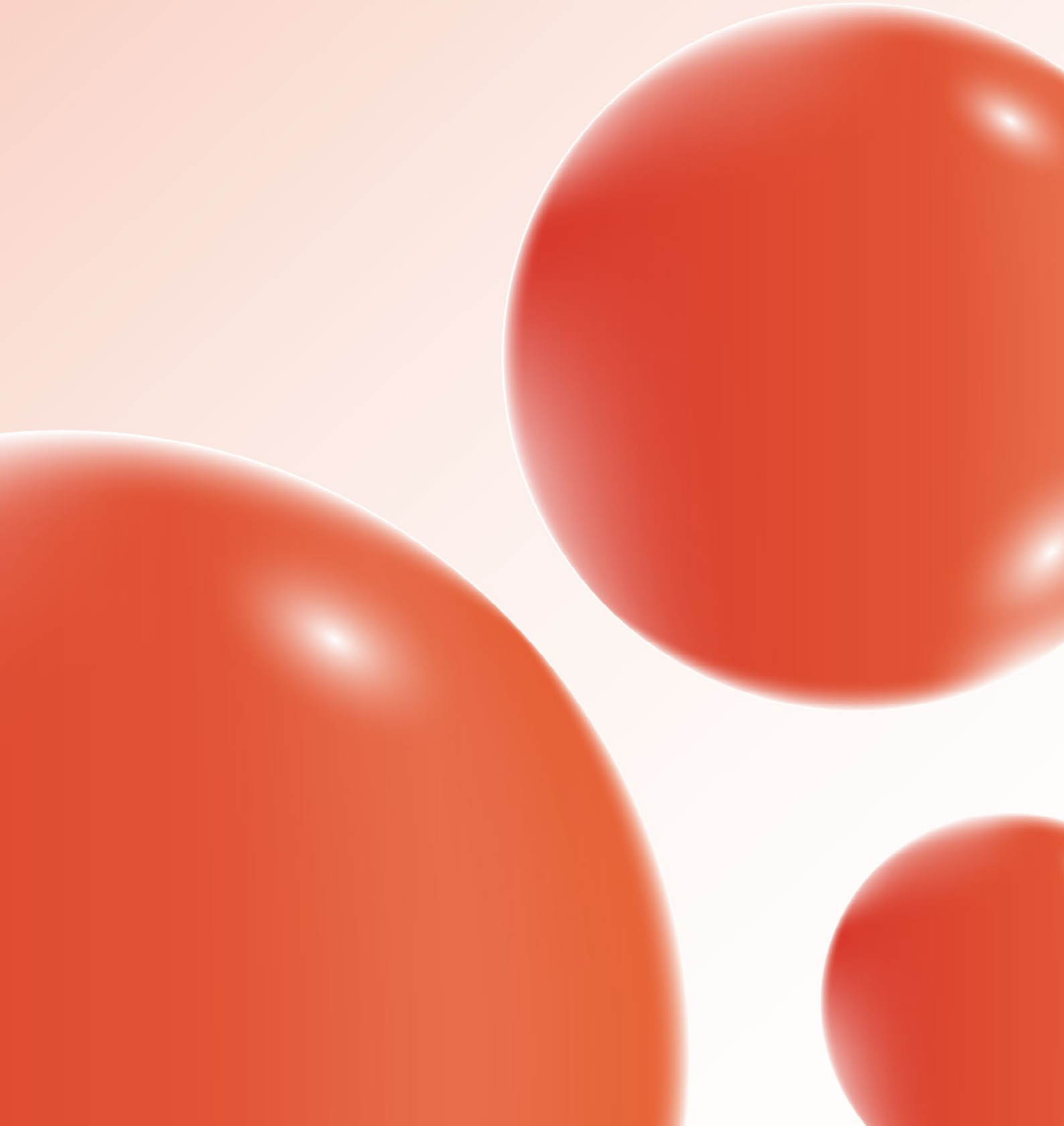




16TH CHALLENGES IN CARDIOLOGY

POSTERS



From Angiography to Physiology: Implementing Quantitative Flow Ratio in Daily Practice

Margarida Figo

UNIDADE LOCAL DE SAÚDE DO ALENTEJO CENTRAL

Introduction

Functional assessment of intermediate coronary lesions remains challenging in daily clinical practice. Fractional Flow Reserve is the reference standard but is underused due to procedural complexity. Quantitative Flow Ratio (QFR) has emerged as an angiography-based alternative for physiological assessment.

Objectives

To describe the initial experience with QFR and its integration into clinical workflow, highlighting its role in decision-making through a representative clinical case.

Material and Methods

A retrospective descriptive analysis of approximately 200 QFR evaluations performed during routine coronary angiography was conducted. QFR analysis was obtained using dedicated software from standard angiographic projections. Workflow feasibility and integration into daily practice were assessed. A representative case of an intermediate coronary lesion with borderline QFR was selected to illustrate clinical application.

Results

QFR analysis was feasible in the majority of cases and was successfully incorporated into routine workflow without significant procedural delay. The technique enabled functional assessment without pressure wire or pharmacological hyperemia. In the presented case, QFR value was 0.81, representing a borderline result. Due to this grey zone finding, invasive physiological assessment was performed to guide final clinical decision-making.

Conclusions

QFR is a practical and feasible tool for functional assessment in routine coronary angiography. Its integration into clinical workflow is achievable and supports decision-making, although borderline values may still require complementary invasive assessment.

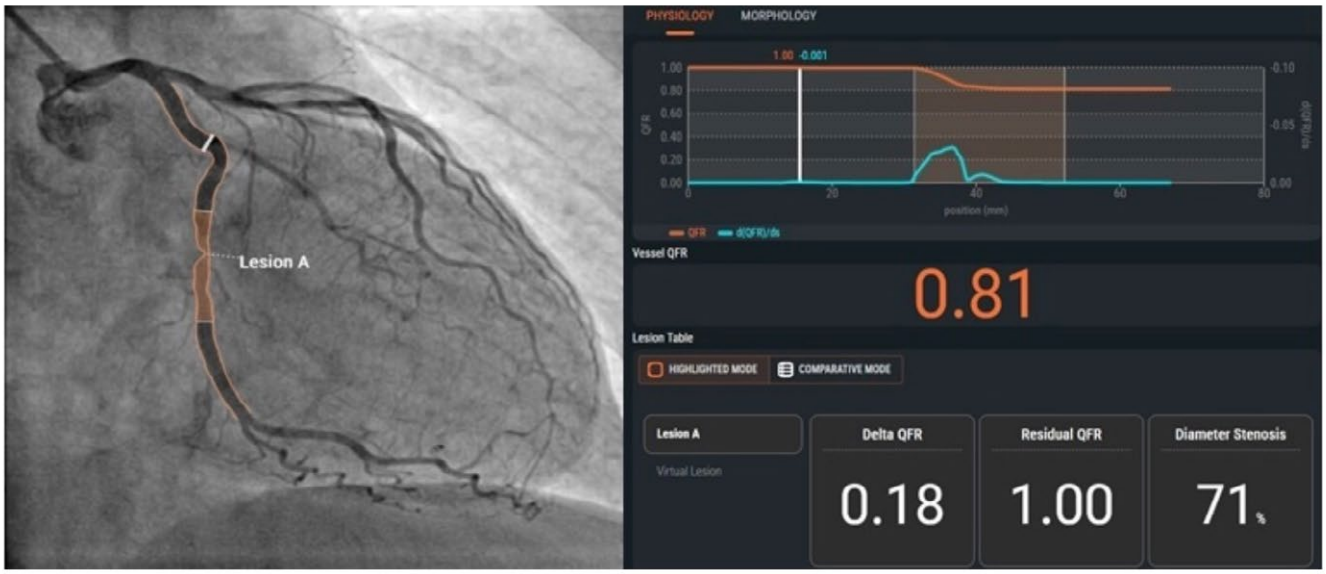


Image 1 - Coronary angiography and QFR analysis demonstrating an intermediate lesion in the left circumflex artery with a borderline QFR value (0.81).

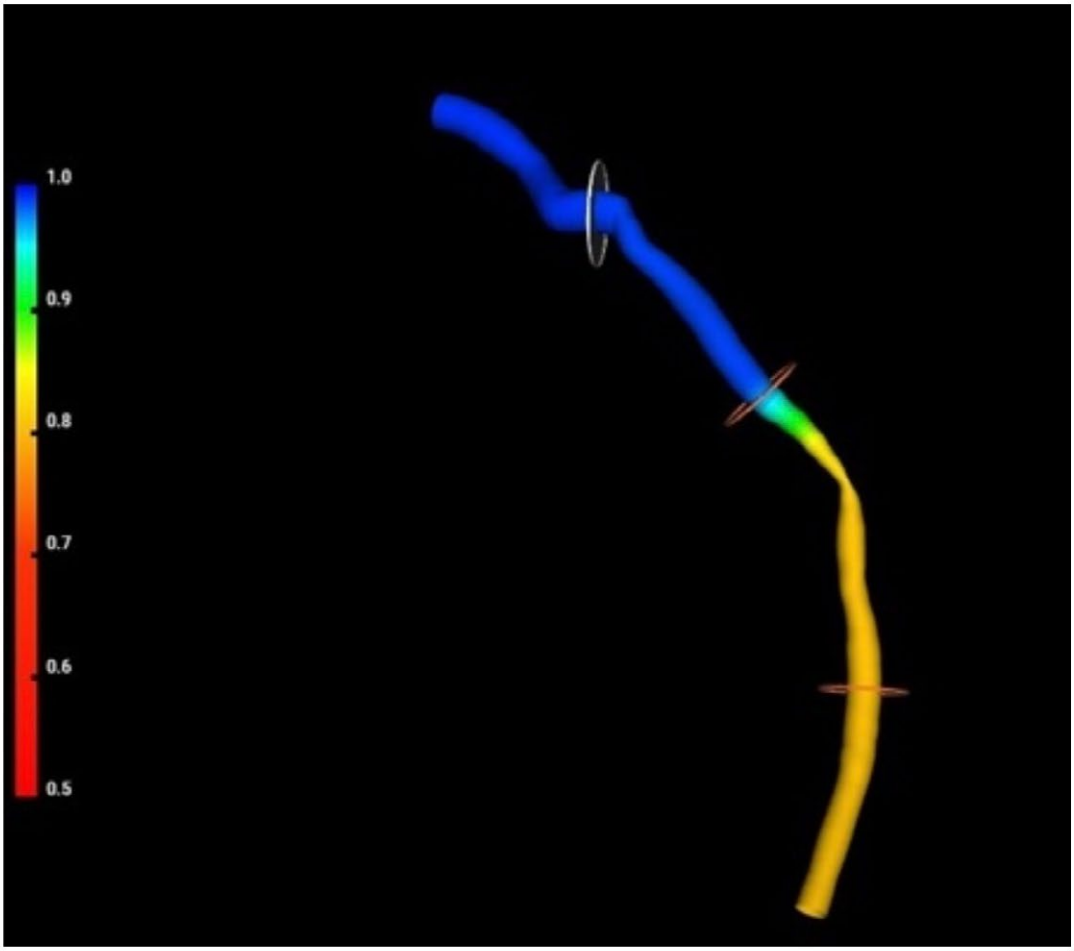


Image 2 - Three-dimensional QFR reconstruction illustrating the functional gradient along the vessel.

Genotype-driven differences in the phenotype and prognosis of patients with hypertrophic cardiomyopathy

André Manuel Martins; Mónica Amado; Joana Pereira; Adriana Vazão; Carolina Esteves; Célia Domingues; Catarina Ruivo; David Durão

HOSPITAL DE SANTO ANDRÉ

Introduction

Hypertrophic cardiomyopathy (HCM) is a heterogeneous autosomal dominant disease commonly caused by pathogenic variants in sarcomere protein genes, although many patients remain genotype-negative or carry variants of uncertain significance (VUS). Despite decades of research, genotype-phenotype correlations remain incompletely understood.

Objectives

To evaluate the influence of genotype on clinical and imaging phenotypes and short-term prognosis in HCM patients followed at a regional Cardiomyopathy Clinic.

Methods

Retrospective, single-center study of patients diagnosed with HCM between 2018 and 2024. Those with uncontrolled hypertension, significant valvular disease, or lacking genetic testing were excluded. Patients were classified into two groups: genotype-negative or VUS (Group 1) and pathogenic (P) or likely pathogenic (LP) (Group 2). Group comparisons and regression analyses identified variables associated with P/LP variants (Table 1A).

Results

Among 145 enrolled patients (61 ± 13 years; 64% male), genetic testing identified P/LP variants in 56 (39%) (Group 2), VUS in 27 (19%), and no detectable mutations in 62 (43%). P/LP carriers more frequently exhibited a midventricular phenotype (82 vs 66%, p=0.038) and had lower rates of overweight (52 vs 69%, p=0.043), hypertension (52 vs 70%, p=0.030), and dyslipidemia (57 vs 76%, p=0.015). This subgroup also had a higher prevalence of family history of HCM (30 vs 6%, p<0.001) and a normal ECG (25 vs 4%, p<0.001). Echocardiography demonstrated greater interventricular septal thickness (17.9 vs 17.4 mm, p=0.046) and left atrial volume index (53.6 vs 50.8 ml/m², p=0.007) in P/LP carriers. hs-TnI levels were likewise higher in this group (17.8 vs 11.3 pg/mL, p=0.032). The median follow-up was 46 months (IQR 35), with no differences between groups in the occurrence of extended MACE (cardiovascular mortality, myocardial infarction, stroke and heart failure hospitalizations) or new-onset AV/IV conduction disturbances or atrial fibrillation. ROC analysis defined cut-offs for continuous variables. In multivariate logistic regression family history of HCM, a normal ECG and hs-TnI ≥ 13.2 pg/ml remained independently associated with P/LP variants (Table 1B).

Conclusions

In this HCM cohort, higher hs-TnI, a normal ECG, and family history of the disease were associated with P/LP variants, while no differences were observed in follow-up outcomes. The small sample size limits conclusions regarding individual gene effects.

Table 1 (A)	Total (n=145)	Group 1 (n=89)	Group 2 (n=56)	p-value
Male gender – n (%)	93 (64)	57 (64)	36 (64)	0.977[a]
Age (years) – mean (SD)	61 (13)	63 (16)	60 (21)	0.157[b]
HCM morphologic subtypes – n (%)				
Apical	27 (19)	24 (27)	3 (5)	**<0.001[a]**
Midventricular	105 (72)	59 (66)	46 (82)	**0.038[a]**
Mixed	13 (9)	6 (4)	7 (13)	0.237[a]
Obstructive HCM – n (%)	55 (38)	33 (37)	22 (39)	0.790[a]
HCM Risk-SCD – median (IQR)	2.62 (1.95)	2.41 (2.01)	3.08 (2.19)	0.105[b]
NYHA Class – n (%)				
NYHA I	92 (63)	57 (64)	35 (63)	0.851[a]
NYHA ≥II	53 (37)	32 (36)	21 (38)	0.851[a]
Mean follow-up (months) – median (IQR)	46 (35)	43 (27)	51 (40)	**0.013[b]**
AV/IV conduction disturbances – n (%)	13 (10)	5 (6)	8 (17)	0.062[a]
New-onset AF – n (%)	13 (9)	10 (11)	3 (5)	0.228[a]
MACE – n (%)	19 (13)	8 (9)	11 (20)	0.064[a]
Past medical history – n (%)				
Overweight[1]	90 (62)	61 (69)	29 (52)	**0.043[a]**
Hypertension	91 (63)	62 (70)	29 (52)	**0.030[a]**
Dyslipidemia	100 (69)	68 (76)	32 (57)	**0.015[a]**
Diabetes mellitus	27 (19)	18 (20)	9 (16)	0.532[a]
Chronic kidney disease[2]	12 (8)	7 (8)	5 (9)	0.821[a]
Atrial fibrillation	26 (18)	12 (14)	14 (25)	0.078[a]
Family history of HCM	22 (15)	5 (6)	17 (30)	**<0.001[a]**
Family history of SCD	15 (10)	6 (7)	9 (16)	0.072[a]
Electrocardiographic parameters – n (%)				
Sinus rhythm	127 (88)	80 (90)	47 (84)	0.289[a]
First-degree AV block	17 (12)	10 (12)	7 (13)	0.834[a]
Right bundle branch block	10 (7)	7 (8)	3 (6)	0.740[a]
Left bundle branch block	8 (6)	5 (6)	3 (6)	0.936[a]
Bifascicular block	8 (6)	6 (7)	2 (4)	0.484[a]
Left ventricular hypertrophy	80 (66)	50 (68)	30 (63)	0.565[a]
T-wave inversion	79 (65)	58 (78)	21 (44)	**<0.001[a]**
Normal ECG	15 (12)	3 (4)	12 (25)	**<0.001[a]**
Echocardiographic parameters				
LV EF (%) – mean (SD)	62.3 (5.6)	62.1 (5.3)	62.4 (5.9)	0.228[c]
LV MWT (mm) – mean (SD)	19.6 (3.7)	19.6 (3.5)	19.5 (3.8)	0.301[c]
IVSd (mm) – median (IQR)	17.4 (3.9)	17.4 (3.8)	17.9 (4.6)	**0.046[b]**
LV mass index (g/m^2) – mean (SD)	164.0 (44.9)	165.2 (45.6)	158.0 (44.1)	0.820[c]
LV EDV index (ml/m^2) – median (IQR)	52.3 (17.9)	51.8 (21.6)	51.8 (17.0)	0.717[b]
RWT – mean (SD)	0.60 (0.14)	0.61 (0.14)	0.58 (0.15)	0.170[c]
TAPSE (mm) – mean (SD)	23.0 (4.1)	22.8 (3.3)	23.4 (3.3)	0.137[c]
E/e' ratio – median (IQR)	12.8 (6.8)	12.6 (5.8)	14.4 (8.4)	0.730[b]
PSAP (mmHg) – median (IQR)	31.0 (10.0)	31.0 (11.0)	32.0 (9.8)	0.786[b]
LV GLS (%) – mean (SD)	-15.4 (3.2)	-15.3 (3.5)	-15.8 (3.1)	0.120[c]
RV GLS (%) – mean (SD)	-20.0 (4.6)	-20.3 (4.0)	-19.8 (5.3)	0.841[c]
LA volume index (ml/m^2) – mean (SD)	52.3 (12.6)	50.8 (11.2)	53.6 (14.8)	**0.007[c]**
LA reservoir strain (%) – median (IQR)	18.0 (11.0)	18.0 (11.0)	18.5 (10.5)	0.904[b]
LA contractile strain (%) – mean (SD)	-9.1 (5.3)	-9.9 (5.0)	-7.8 (5.8)	0.318[c]
LA conduit strain (%) – median (IQR)	-9.0 (6.0)	-8.0 (7.0)	-10.0 (6.5)	0.264[b]
Analytical parameters				
NT-proBNP (pg/ml) – median (IQR)	662.0 (1029.0)	599.0 (1041.0)	839.0 (1032.0)	0.172[b]
hs-TnI (pg/ml) – median (IQR)	14.8 (17.6)	11.3 (20.1)	17.8 (14.4)	**0.032[b]**
Creatinine (mg/dl) – median (IQR)	0.87 (0.35)	0.87 (0.34)	0.86 (0.39)	0.730[b]

Table 1 (A). Patient baseline characteristics. Statistical analysis: [a]Chi-square test, [b]Mann-Whitney U test, [c]t-student test. [1]BMI ≥ 25 kg/m2, [2]GFR < 60 ml/min/1.73m2 (2021 CKD-EPI). **AF** – atrial fibrillation, **AV** – atrioventricular, **EDV** – end diastolic volume, **E/e'** – E-wave/e'-wave, **EF** – ejection fraction, **GLS** – global longitudinal strain, **HCM** – hypertrophic cardiomyopathy, **hs-TnI** – high-sensitivity troponin I, **IV** – intraventricular, **IVSd** – interventricular septum thickness end diastole, **LA** – left atrium, **LV** – left ventricle, **MACE** – Major Adverse Cardiac Events (cardiovascular mortality, myocardial infarction, stroke and heart failure hospitalizations), **MWT** – maximal wall thickness, **PSAP** – pulmonary artery systolic pressure, **RV** – right ventricle, **RWT** – relative wall thickness, **TAPSE** – tricuspid annular plane systolic excursion, **SCD** – sudden cardiac death

Table 1 (B)

Variables	OR	CI 95%	p-value
Midventricular subtype	1.968	0.639 – 6.059	0.238
Family history of HCM	5.904	1.363 – 25.581	**0.018**
Normal ECG	6.392	1.336 – 30.574	**0.020**
IVSd ≥ 17.3 mm	1.776	0.671 – 4.701	0.247
LA volume index ≥ 54.3 ml/m^2	1.798	0.703 – 4.601	0.221
hs-TnI ≥ 13.2 pg/ml	2.687	1.009 – 7.153	**0.048**

Table 1 (B). Multivariate logistic regression

Predictors of Pacemaker Implantation in transthyretin amyloid cardiomyopathy

André Manuel Martins; Joana Pereira; Mónica Amado; Adriana Vazão; Carolina Esteves; Catarina Ruivo; Célia Domingues; David Durão

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Introduction

Transthyretin cardiac amyloidosis (ATTR-CM) is increasingly recognized as an important cause of heart failure (HF). Conduction system involvement is common and largely driven by progressive extracellular amyloid fibril deposition, with approximately 9.5% of patients ultimately requiring permanent pacemaker implantation. However, reliable risk stratification for device implantation in this population remains limited.

Objectives

To identify predictors of pacemaker implantation in patients with ATTR-CM followed at a Cardiomyopathy Clinic in a regional hospital in Portugal.

Methods

Retrospective single-center study of patients diagnosed with wild type ATTR-CM according to theESC algorithm between 2018 and 2024. Data on clinical, electrocardiographic and echocardiographic parameters were collected at the time of diagnosis (Table 1A). The primary endpoint was the occurrence of permanent pacemaker implantation during follow-up. Patients who underwent device implantation (Group 1) were compared with those who did not (Group 2).

Results

A total of 72 patients were included; 93% were male, with a median age of 81 years (IQR 8). Fifteen patients (21%) underwent permanent pacemaker implantation (Group 1). The main indications for device implantation were complete atrioventricular (AV) block (n=6), bradyarrhythmia due to atrial fibrillation (n=5), trifascicular block (n=2), second-degree Mobitz 2 AV block (n=1), and sinus node disease (n=1). Regarding clinical characteristics, age was higher in Group 1 (84 vs. 80 years, p=0.048), with no differences observed for other clinical variables. For electrocardiographic parameters, only QRS duration differed significantly, being longer in Group 1 (151 vs. 107 ms, p<0.001). Echocardiographically, patients in Group 1 exhibited lower left ventricular (LV) global longitudinal strain (GLS) (-8.1 vs. -11.0%, p=0.033) and right ventricular (RV) GLS (-8.2 vs. -12.8%, p=0.004). ROC curve analysis defined optimal cut-offs for continuous variables. In multivariate analysis, only age and QRS duration remained independently associated with pacemaker implantation, with a non-significant trend for higher risk in patients with more impaired LV GLS (Table 1B).

Conclusions

In this cohort, permanent pacemaker implantation occurred in approximately one-fifth of ATTR-CM patients. Older age and prolonged QRS duration were independent predictors of device implantation, while echocardiographic strain showed no independent association. These findings underscore the importance of routine electrocardiographic assessment in the risk stratification of patients with ATTR-CM.

Table 1 (A)

	Total (n=72)	Group 1 (n=15)	Group 2 (n=57)	p-value
Male gender – n (%)	67 (93)	14 (93)	53 (93)	0.962[a]
Age at diagnosis (years) – median (IQR)	81 (8)	84 (6)	80 (6)	**0.048**[b]
NYHA Class				
NYHA I	9 (13)	2 (13)	7 (12)	0.913[a]
NYHA II	52 (72)	9 (60)	43 (75)	0.235[a]
NYHA III	11 (15)	4 (27)	7 (12)	0.224[a]
Mean follow-up (months) – mean (SD)	33 (16)	40 (20)	32 (15)	0.073[c]
Past medical history – n (%)				
Hypertension	54 (75)	14 (93)	40 (70)	0.065[a]
Dyslipidemia	55 (76)	12 (80)	43 (75)	0.711[a]
Atrial fibrillation	47 (65)	10 (67)	37 (65)	0.899[a]
Diabetes mellitus	30 (42)	9 (60)	21 (37)	0.106[a]
Chronic kidney disease	36 (50)	7 (47)	29 (51)	0.772[a]
Anemia	20 (28)	6 (40)	14 (25)	0.235[a]
Coronary artery disease	13 (18)	2 (13)	11 (19)	0.723[a]
Valvular heart disease	18 (25)	4 (27)	14 (25)	0.867[a]
Electrocardiographic parameters				
First degree AV-Block – n (%)	18 (25)	4 (27)	14 (25)	0.867[a]
RBBB – n (%)	4 (6)	1 (7)	3 (5)	1.000[a]
LBBB – n (%)	11 (15)	4 (27)	7 (12)	0.224[a]
Bifascicular block – n (%)	28 (39)	7 (47)	21 (37)	0.487[a]
QRS duration (ms) – mean (SD)	119 (29)	151 (29)	107 (35)	**<0.001**[c]
Echocardiographic parameters				
LVEF (%) – median (IQR)	54 (12)	52 (10)	56 (14)	0.238[b]
IVSd (mm) – mean (SD)	18.2 (3.2)	18.1 (2.5)	18.2 (3.3)	0.382[c]
LV mass index (g/m^2) – median (IQR)	178.3 (64.8)	189.6 (92.04)	177.7 (58.7)	0.798[b]
LA volume index (ml/m^2) – mean (SD)	59.4 (13.4)	63.0 (13.2)	58.7 (13.4)	0.182[c]
E/e' – median (IQR)	16.0 (6.3)	17.7 (8.9)	15.7 (6.4)	0.270[b]
LA reservoir strain (%) – median (IQR)	7.0 (8.0)	4.0 (4.0)	7.0 (8.0)	0.237[b]
LV GLS (%) – mean (SD)	-10.5 (3.4)	-8.1 (3.2)	-11.0 (3.2)	**0.033**[c]
RV GLS (%) – mean (SD)	-12.1 (4.6)	-8.2 (3.9)	-12.8 (4.4)	**0.004**[c]
PSAP/TAPSE (mm/mmHg) – mean (SD)	0.49 (0.15)	0.41 (0.09)	0.51 (0.15)	0.194[c]

Table 1 (B)

Variables	OR	CI 95%	p-value
Age ≥ 83 years	8.146	1.233-53.823	**0.029**
LV GLS ≥ -10.9%	9.989	0.776-128.5	0.077
RV GLS ≥ -11.4%	3.444	0.509-23.310	0.205
QRS duration ≥ 121.5ms	14.521	1.948-108.242	**<0.001**

Table 1. Patient baseline characteristics (A) and multivariate logistic regression (B). Statistical analysis: [a]Chi-square test, [b]Mann-Whitney U test, [c]t-student test. Abbreviations: **AV** – atrioventricular, **CI** – confidence interval, **E/e'** – E-wave/e'-wave, **IQR** – interquartile range, **IVSd** – interventricular septum thickness end diastole, **GLS** – global longitudinal strain, **LA** – left atrial, **LBBB** – left bundle branch block, **LV** – left ventricle, **LVEF** – left ventricular ejection fraction, **OR** - odds ratio, **PSAP** - pulmonary artery systolic pressure, **RBBB** – right bundle branch block, **RV** – right ventricle, **SD** – standard deviation, **TAPSE** – tricuspid annular plane systolic excursion.

Blood Pressure Outcomes After Renal Denervation: A Single-Center Real-World Experience

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Introduction

Renal sympathetic overactivity plays a key role in the pathophysiology of hypertension, and renal denervation (RDN) has gained increasing interest as an adjunct or alternative to antihypertensive pharmacotherapy. However, the magnitude of blood pressure (BP) reduction after RDN varies considerably across patients, and identifying procedural predictors of treatment response remains an important unmet need.

Aim

To evaluate whether patients undergoing RDN experienced a significant improvement in BP and to determine whether the magnitude of this improvement was associated with the total number of ablation applications.

Material and Methods

A single-center retrospective analysis was performed in patients treated with RDN. BP values before and after the procedure were compared using paired-samples t-tests. Pearson correlation analyses were conducted to assess the association between the number of ablation applications and BP reduction.

Results

Twenty-five patients were included (mean age 61 ± 9 years, 64% male), with 88% treated with four or more antihypertensive agents. Baseline characteristics are shown in Table 1.

The mean total number of ablation applications was 29 ± 8, with 15 ± 4 delivered to the left renal artery and 14 ± 5 to the right renal artery. RDN resulted in statistically significant reductions in systolic BP (154.6 ± 18.6 vs. 135.2 ± 19.1 mmHg; $p < 0.001$), diastolic BP (85.6 ± 14.0 vs. 76.4 ± 13.6 mmHg; $p = 0.001$), and mean BP (108.6 ± 13.4 vs. 96.5 ± 13.3 mmHg; $p < 0.001$).

A moderate, statistically significant negative correlation was observed between the number of left renal artery applications and reductions in systolic BP ($r(15) = -0.59$, $p = 0.014$) and mean BP ($r(15) = -0.49$, $p = 0.046$), indicating that a higher number of left-sided applications was associated with greater BP improvement. However, no significant association was found with the percentage of impedance drop in the left renal artery applications ($p = 0.500$) or with the maximum temperature reached ($p = 0.342$).

No intra- or post-procedural complications occurred.

Conclusion

In this single-center real-world cohort, RDN produced significant reductions in systolic, diastolic, and mean BP, supporting its therapeutic value in resistant hypertension. The findings suggest that greater ablation intensity in the left renal artery may enhance treatment response; however, larger studies are needed to clarify the role of application number and distribution as predictors of clinical outcomes.

	n = 25
Age, mean ± SD, years	61.3 ± 9.2
Male, n (%)	16 (64)
Dyslipidemia, n (%)	17 (68)
Type 2 Diabetes Mellitus, n (%)	13 (52)
Obesity, n (%)	15 (60)
BMI, mean ± SD	31.6 ± 4.7
OSA, n (%)	10 (40)
Smoker, n (%)	6 (24)
CKD, n (%)	10 (40)
CAD, n (%)	8 (32)
CVD, n (%)	4 (16)
PAD, n (%)	2 (8)
ACEi/ARB, n (%)	25 (100)
CCB, n (%)	23 (92)
Betablocker, n (%)	21 (84)
Diuretic, n (%)	16 (64)
Methyldopa, n (%)	3 (12)
Rilmenidine, n (%)	16 (64)
≥4 drug classes, n (%)	22 (88)

Footnote: ACEi - Angiotensin-converting enzyme inhibitors. ARB - Angiotensin receptor blockers. BMI - Body mass index. CAD - Coronary artery disease. CCB - Calcium Channel Blocker. CKD - Chronic Kidney Disease. CVD - Cerebrocascular Disease. OSA - Obstructive Sleep Apnea. PAD - Peripheral Artery Disease. IQR - Interquartile Range. SD - Standard deviation.

Table 1 Patient Baseline Characteristics

Mind the [knowledge] gap: cardiovascular disease awareness and literacy in postmenopausal portuguese women

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Introduction

Cardiovascular disease (CVD) is frequently underdiagnosed in women and remains the leading cause of death in this population, with increased risk following menopause. Understanding the level of knowledge and awareness regarding CVD and cardiovascular risk factors (CVRF) in postmenopausal women is crucial for developing effective prevention and management strategies.

Purpose

This study aimed to assess cardiovascular health literacy in a Portuguese postmenopausal population.

Material and Methods

We conducted a cross-sectional survey with postmenopausal women recruited during a cardiovascular screening event in Portugal (May 2023). Participants completed an adapted validated questionnaire evaluating awareness of CVD risk, recognition of symptoms and signs, as well as knowledge regarding the causes, prevention, and appropriate response to CVD. Statistical analyses were conducted with appropriate tests for data distribution.

Results

We included 127 post menopausal women with a mean age of 65±9 years. The most common CVRF was hypertension (55.3%), followed by dyslipidaemia (13.0%) and diabetes (13.0%). Only 11.4% were current smokers, and 36.6% reported daily alcohol consumption. Economic issues were the most frequently cited barrier to maintaining a healthy lifestyle (47.5%), followed by the perception of already having a healthy lifestyle (43.3%) and lack of time (29.2%).

21.5% had a Bachelor's degree or higher education level and the majority resided in urban areas (92.1%).

Concerning CVD literacy, most women (75.2%) found it easy to obtain information about CVD in women and 67.8% reported hearing about CVD within the past year. However, only 14% felt well informed on the topic. Awareness of CVD was limited, with 68.3% of participants reporting being unaware or having little awareness.

When assessing knowledge about CVD in women, only 36.5% could correctly identify acute myocardial infarction symptoms. Although 67.5% were aware of the increased CVD risk associated with the postmenopausal period, only 27.2% recognized CVD as the leading cause of death among women, and only 26.9% acknowledged worse cardiovascular outcomes in women (see Table 1). Lower awareness of CVD in women was significantly associated with lower educational levels ($p = 0.026$) and perceived difficulty in obtaining information about CVD in women ($p = 0.001$). Conversely, greater awareness of CVD was linked to better knowledge of CVD prevention ($p = 0.025$) and of cardiovascular risk factors ($p = 0.013$).

Conclusion

Our study underscores the need to enhance CVD literacy and awareness among postmenopausal women, particularly in those with lower education levels. Targeted interventions addressing these knowledge gaps could play a crucial role in empowering women and improving awareness and health outcomes.

Table 1 – Characteristics of the study population, Cardiovascular Health Education and Literacy

	n = 127
Patient Demographics and CVRF	
Age, mean (±SD), years	65±9
Urbanization Level of Residence, n (%)	
Urban	116 (92.1)
Rural	10 (7.9)
Education Level, n (%)	
Primary Education	36 (29.8)
Secondary Education	59 (48.8)
Bachelor or Superior	26 (21.5)
Cardiovascular risk factors, n (%)	
Hypertension	55 (55.3)
Dyslipidaemia	25 (13.0)
Diabetes mellitus	16 (13.0)
Smoking History, n (%)	
Never Smoked	88 (71.5)
Former Smoker (quitted >1year)	18 (14.6)
Former Smoker (quitted <1year)	3 (2.4)
Current smoker	14 (11.4)
Alcohol consumption, n (%)	
Never drinks alcohol	66 (63.5)
1-2 glasses a day	32 (30.8)
≥3 glasses a day	6 (5.8)
More than 2 times a week	14 (31.1)
Cerebrovascular disease, n (%)	3 (2.4)
Cardiovascular disease, n (%)	9 (7.3)
Cardiovascular Health Education	
Degree of information received about CVD in women, n (%)	
I was well informed	17 (14.0)
I received some information	31 (25.6)
I received little information	32 (26.4)
I was not informed	41 (33.9)
Self-evaluation of knowledge about CVD health literacy, n (%)	
I know very well	5 (4.2)
I know well	33 (27.5)
I know little	58 (48.3)
I do not know	24 (20.0)
Finds it easy to access information about CVD in women, n (%)	61 (75.2)
Heard about CVD in women in the last year, n (%)	
Feels comfortable discussing their illness with the doctor, n (%)	76 (91.6)
Barriers to a healthy lifestyle, n (%)	
Economic issues	57 (47.5)
Already has a healthy lifestyle	52 (43.3)
Lack of time	35 (29.2)
Does not believe in CVD prevention	11 (9.2)
Lack of information	13 (10.8)
Not interested	5 (4.2)
Others	16 (13.4)
Cardiovascular Health Literacy	
Cardiovascular Disease in general, n (%)	
Recognizes the main cause of death among women	34 (27.2)
Recognizes the most prevalent disease in women	20 (16.4)
Links menopause to heightened risk of CVD	83 (67.5)
Links depression to heightened risk of CVD	94 (76.4)
Acknowledges worse cardiovascular outcomes in women	32 (26.9)
Acute Myocardial Infarction, n (%)	
Correctly identifies Acute Myocardial infarction symptoms	46 (36.5)
Recognizes angina and equivalents	110 (86.8)
Knows how to act in Acute Myocardial Infarction	75 (65.2)
Stroke, n (%)	
Correctly identifies stroke symptoms	64 (50.8)
Heart Failure, n (%)	
Correctly identifies Heart failure symptoms	49 (38.9)
Level of knowledge about CVD prevention[1], n (%)	
Low	28 (22.8)
Medium	16 (13.0)
High	79 (64.2)
Level of knowledge about CVFR[1], (n%)	
Low	30 (24.6)
Medium	60 (49.2)
High	32 (26.2)

Footnote: CVD – Cardiovascular Disease; CVFR – Cardiovascular Risk Factors; [1]low: <50% correct answers, medium: 50-70% correct answers, high: >70% correct answers

Procedural efficiency of pulsed-field ablation compared with high-power short-duration radiofrequency ablation in a real-world medium-volume centre

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Introduction

Atrial fibrillation (AF) is a major contributor to morbidity and healthcare resource utilization. Increasing disease burden and prolonged waiting lists continue to limit timely access to catheter ablation. Pulsed-field ablation (PFA) has recently emerged as a non-thermal modality enabling rapid, tissue-selective lesion formation, while high-power short-duration radiofrequency ablation (RF-HPSD) represents an optimization of conventional thermal techniques.

Purpose

This study aimed to compare procedural efficiency and arrhythmia recurrence between PFA and RF-HPSD in a medium-volume centre.

Material and Methods

We included 112 consecutive patients who underwent AF ablation between December 2023 and May 2025. Baseline characteristics, procedural metrics, fluoroscopy parameters, and recurrence outcomes were collected. AF recurrence was assessed using Kaplan-Meier analysis and Cox proportional hazards modelling.

Results

The mean age was 65 ± 11 years; 58% were male. Of all procedures, 27.7% were performed using PFA with a pentaspline catheter and 72.3% using RF-HPSD. Redo procedures accounted for 24.1%. Baseline characteristics are shown in Table 1. The overall complication rate was 2.7%, all in the HPSD group.

AF recurrence occurred in 15.3% of patients overall (9.7% with PFA vs 17.5% with RF-HPSD) after a mean follow-up of 293.6 ± 175.0 days. Mean fluoroscopy time was significantly higher with PFA (15.1 ± 10.4 vs 9.4 ± 6.0 min; p = 0.006), although radiation dose did not differ significantly (PFA: 53.4 mGy/m² [IQR 22.3-84.8] vs RF-HPSD: 31.5 mGy/m² [IQR 18.5-56.6]; p = 0.09). In first-time (non-redo) procedures, fluoroscopy time remained higher with PFA (14.1 ± 18.8 vs 8.8 ± 5.4 min; p = 0.007).

Despite this, median total procedural time (skin-to-skin) was significantly shorter with PFA (64 min [IQR 46-96] vs 85 min [IQR 75-97]; p = 0.009). Kaplan-Meier analysis showed longer arrhythmia-free survival with PFA (log-rank p = 0.03). In the Cox model, RF-HPSD was associated with a higher recurrence risk (HR 4.80; p = 0.047).

Conclusions

In this medium-volume centre, PFA provided superior procedural efficiency, reducing total procedure time by approximately 25% compared with RF-HPSD, despite increased fluoroscopy time and similar radiation exposure. PFA was also associated with longer arrhythmia-free survival. These findings suggest that incorporation of PFA may confer meaningful operational benefits, potentially reducing ablation-related waiting list burden by up to one quarter.

	PFA (n=31)	RF - HPSD (n=81)	p value
Age, mean ± SD, years	65.3 ± 11.0	64.5 ± 11	0.721
Male, n (%)	16 (51.6)	49 (60.5)	0.394
Hypertension, n (%)	15 (48.4)	51 (63.7)	0.139
Dyslipidemia, n (%)	14 (45.2)	41 (51.2)	0.331
Type 2 Diabetes Mellitus, n (%)	4 (12.9)	14 (17.4)	0.556
Obesity, n (%)	6 (19.4)	22 (28.2)	0.340
OSA, n (%)	1 (3.2)	23 (28.7)	**0.003**
LVEF, median (IQR), %	59.9 (55.0 - 63.0)	60.0 (53.5 - 66.0)	0.872
LAVi, mean ± SD,mL/m²	41.7± 11.7	40.9 ± 10.4	0.742
LA AP diameter, mean ± SD, mm	42.9 ± 6.4	42.5 ±11.2	0.875
Redo procedures, n (%)	4 (12.9)	23 (28.4)	0.086
Paroxysmal AF n (%)	24 (77.4)	54 (69.2)	0.638
Persistent AF, n (%)	4 (12.9)	16 (20.5)	0.346
Long-standing persistent AF, n (%)	3 (9.7)	16 (20.5)	0.813
Fluoroscopy time, mean ± SD, min	15.1 ± 10.4	9.4 ± 6.0	**0.006**
Total procedural time, median (IQR), min	64 (46 - 96)	85 (75 - 97)	**0.009**
Fluoroscopy dose, median (IQR), mGy	75.7 (43.5 - 126.3)	55.1 (29.2 - 101.1)	0.090
Fluoroscopy dose indexed, median (IQR), mG	53.4 (22.3 - 84.8)	31.5 (18.5 - 56.6)	0.169
Complications, n (%)	0 (0)	3 (3.7)	0.559
Recurrence, n (%)	3 (9.7)	14 (17.5)	0.389

Footnote: PFA - Pulsed-Field Ablation. RF-HPSH - High-Power Short-Duration Radiofrequency Ablation. OSA - Obstructive Sleep Apnea. LVEF - Left Ventricular Ejection Fraction. LAVi - indexed Left Atrial Volume. LA - left atrial. AP - anteroposterior. AF - Atrial Fibrillation
IQR - Interquartile Range. SD - Standard deviation.

Table 1 Comparison of baseline characteristics and procedural results between PFA and RF-HPSD.

Cardiovascular Risk after Acute Myocardial Infarction: Impact of LDL Cholesterol and Triglycerides Control in Secondary Prevention

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Background

After an acute myocardial infarction (AMI), many patients continue to develop cardiovascular events, a phenomenon known as residual risk. This is due, among other factors, to suboptimal control of lipid parameters. This study aimed to characterize the control of low-density lipoprotein cholesterol (LDL-C) and Triglycerides after AMI, testing the hypothesis that their adequate control reduces the risk of subsequent cardiovascular events.

Methods

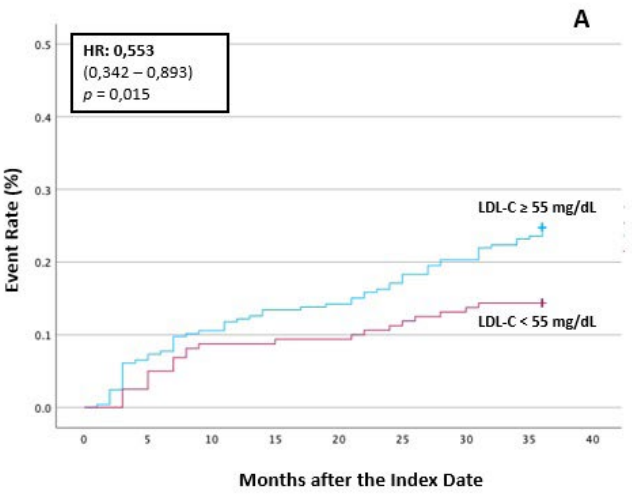
An observational, cohort, retrospective study was developed based on the analysis of clinical records of AMI cases, between 2020 and 2022. The evolution and control of LDL-C and Triglycerides were characterized at a follow-up visit (3-12 months after AMI), and the incidence of cardiovascular events and death was recorded over a 3-year follow-up period. The association between lipid parameters and the occurrence of adverse events was assessed using Cox regressions.

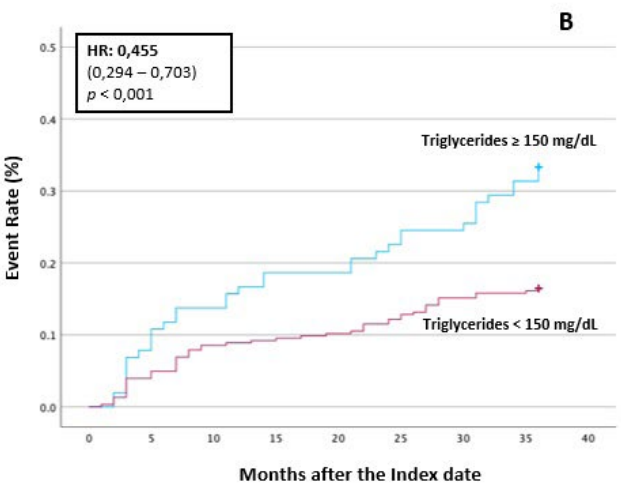
Results

A final sample of 406 patients was included, consisting of 324 men (79.8%) and 82 women (20.2%), with a mean age of 63±11.8 years. 39.4% of patients achieved LDL-C (<55 mg/dL) and 74.9% achieved Triglycerides (<150 mg/dL) control. Combination therapy with statin and ezetimibe was associated with better LDL-C control compared to statin monotherapy (p=0.004). During the follow-up period, 20.7% of patients developed at least one adverse event. LDL-C control (Hazard ratio (HR)=0.526; 95% confidence interval (CI): 0.320-0.866; p=0.012) and Triglycerides control (HR=0.607; 95% CI: 0.374-0.984; p=0.043) were associated with a lower risk of adverse events in adjusted analyses. Isolated Triglycerides control (Triglycerides <150 mg/dL and LDL-C ≥55 mg/dL) maintained the protective effect observed (HR=0.486; 95% CI: 0.280-0.842; p=0.010).

Conclusions

Adequate control of LDL-C and Triglycerides after AMI reduced the risk of cardiovascular events. Triglycerides demonstrated value as an independent marker of residual risk, representing an additional opportunity for intervention in secondary prevention. However, the achievement of therapeutic targets remains suboptimal, reflecting the persistence of significant residual lipid risk after AMI.


A
HR: 0,553
(0,342 – 0,893)
p = 0,015
LDL-C ≥ 55 mg/dL
LDL-C < 55 mg/dL
Event Rate (%)
Months after the Index Date


B
HR: 0,455
(0,294 – 0,703)
p < 0,001
Triglycerides ≥ 150 mg/dL
Triglycerides < 150 mg/dL
Event Rate (%)
Months after the Index date

Impact of Cisplatin Instillation on Pericardial Effusions of Malignant Etiology

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Background

Malignant pericardial effusion represents a manifestation of advanced oncologic disease, associated with high morbidity and recurrence rates after drainage. Intrapericardial instillation of cytotoxic agents, such as cisplatin, has been proposed as an adjunct strategy to reduce recurrence, although robust evidence remains limited.

Objectives

To evaluate the efficacy and safety of intrapericardial cisplatin instillation in neoplastic pericardial effusions.

Methods

This retrospective 6-year study (2019-2024) included 35 patients with malignant pericardial effusion, allocated into two groups: control (prolonged drainage; n=12) and intervention (cisplatin instillation following prolonged drainage; n=23). Clinical, echocardiographic, analytical and cytological data were collected, as well as information regarding effusion recurrence and procedure-related complications.

Results

Baseline characteristics were similar between groups. Most patients had advanced disease (≥80% in stage IV). Lung adenocarcinoma was the predominant malignancy, in which EGFR mutations were the most frequently identified. The complication rate during hospitalization was 16.7% in the control group (two events: acute pulmonary edema and hypotension) and 34.8% in the intervention group (arrhythmias 26.1%; febrile syndrome 17.4%). One patient in the control group developed constrictive pericarditis as a late complication. Recurrence occurred in 8.3% of patients in the control group versus 17.4% in the intervention group. No significant differences were observed between groups regarding complication or recurrence rates.

Conclusions

Intrapericardial cisplatin instillation demonstrated an overall acceptable safety profile but did not show clear superiority over prolonged drainage alone in preventing recurrence. The prevalence of EGFR mutations and the tendency toward higher recurrence in these patients suggest a potential contribution of molecular factors to effusion behavior. These findings reinforce the need for larger prospective studies to clarify these results and optimize therapeutic strategies.

CA125 - a promising marker for sub-clinical LV disfunction in AMI patients

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Introduction

Cancer antigen 125 (CA125) is a tumour marker of epithelial ovarian cancer. Increasing evidence demonstrates that it also reflects pathophysiological processes beyond malignancy, for instance, as a biomarker in several cardiovascular conditions, particularly congestion and neurohormonal activation. However, the role of this marker in acute myocardial infarction (AMI) patients is not well established. Understanding whether CA-125 has prognostic value in AMI could help refine early risk stratification, in addition to traditional biomarkers. Motivated by these potential clinical applications, our center incorporated CA-125 into routine laboratory panel obtained upon admission for AMI.

Aim

To evaluate whether CA125 levels on admission are associated with left ventricular systolic function, assessed by GLS and LVEF, in patients with acute myocardial infarction.

Methods

We conducted a single-center, observational, retrospective study, including consecutive patients admitted with AMI between July 2025 and October 2025. CA125 was added to standard admission laboratory profile, allowing systematic measurement within 24h after admission.

Results and Discussion

A total of 52 patients were included, consisting of 38 (73,1%) men and 14 (26,9%) women with a median age of 67 (54-77) years. Patients were followed throughout hospitalization for the assessment of in-hospital outcomes. The primary endpoint was the evaluation of left ventricular (LV) function, assessed through ejection fraction (EF) and global longitudinal strain (GLS) measured by transthoracic echocardiography. Acquisition and interpretation of echocardiographic parameters followed current guideline-recommended standards.

We aimed to assess a possible association between CA125 levels and both GLS and left ventricular ejection fraction (LVEF) after AMI. CA125 values did not follow a normal distribution on normality testing; therefore, a Spearman's correlation was applied. Higher CA125 levels were moderately associated with worse GLS (Spearman's rho=0.468, p==0.012), whereas there was no significant correlation between CA125 and LVEF (Spearman's rho=−0.202, p =0.151).

Conclusion

GLS is a sensitive marker of subclinical LV systolic dysfunction, detecting early impairment even when LVEF remains preserved. In this cohort of AMI patients, the moderate association between hight CA125 levels and worse GLS, in the absence of a significant correlation with LVEF, suggests that CA125 is linked to subclinical LV systolic dysfunction. These findings support the use of CA125 as a surrogate marker of prognosis and risk stratification in post-infarction patients. Large prospective studies are warranted to confirm whether integrating GLS and CA125 can improve prediction of adverse outcomes and guide follow-up intensity and therapeutic decisions.

VARIABLES	FREQUENCY
Sex	Male N= 38 (73,1%) | Female N= 14 (26,9%)
Idade	Median 67 (54-77)
BMI	Median 27 (25-28)
Hypertension	n = 37 [71,2%]
Dyslipidemia	n = 36 [79,2%]
Diabetes	n = 20 [38,5%]
Smoking	n = 20 [38,5%]
ACS	STEMI n = 20 | NSTEMI n = 32
Killip Kimbal upon admission	KK I n = 41 | KK II n= 8 | KK III n= 2 | KK IV n= 1
LVEF	Median 55% (40% - 55%)
LV GLS	Median -15.6% (-18.7% - -9.7%)

Sex-based differences between pre-intervention outcomes in patients with severe aortic stenosis - a real-world study

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Introduction

Transcatheter aortic valve implantation (TAVI) is the preferred treatment for severe aortic stenosis (SAS) in older patients (pts), despite limited availability and usually long waiting times. Despite similar incidences between sexes, underlying pathophysiological mechanisms may differ. Identifying sex-related differences in pre-procedural outcomes may improve risk stratification, influence procedural aspects and enhance prognosis.

Objectives

To characterize and compare pre-procedural outcomes between sexes of SAS pts awaiting TAVI.

Methods

Single-center retrospective cohort study of SAS pts who consecutively underwent pre-TAVI cardiac computed tomography (CCT) protocol between June 2022 and October 2025. Demographic and clinical data, transthoracic echocardiography (TTE), coronary angiography and CCT parameters were collected. The pre-procedural endpoint was a composite of cardiovascular (CV) hospitalization, all-cause mortality, and major adverse cardiovascular events (MACE), consisting of CV mortality, non-fatal acute myocardial infarction and stroke, whichever occurred first. Male (group A) and female (group B) pts were compared using SPSS v30.

Results

Overall, 240 pts (mean age 81.7±5.3 years) underwent pre-TAVI CCT, consisting of 121 males (group A) and 119 females (group B). Excepting smoking, history of percutaneous coronary intervention and obstructive coronary artery (CAD) disease, which were more frequent in males (15.7 vs 0.8%, $p<0.001$; 14.0% vs 5.0%, $p=0.018$; 42.4 vs 20.7%, $p<0.001$), demographic or clinical characteristics did not differ (table 1). Females presented higher left ventricular (LV) ejection fraction on both CCT (60.2 vs 67.4%, $p=0.017$) and TTE (58.0 vs 62.3%, $p<0.001$), smaller aortic valve area (AVA) ($p<0.001$) and thinner septal ($p=0.007$) and posterior wall ($p=0.010$) thicknesses on TTE. At a mean follow-up of 9.9 months, no differences were observed in pre-procedural outcomes (32.5 vs 26.5%, $p=0.311$) and any of its components, including CV hospitalization (table 1).

Conclusions

In this cohort of SAS pts awaiting TAVI, sex-related differences were observed regarding the prevalence of smoking and obstructive CAD (diagnosed and intervened), as well as in AVA and LV geometry and function assessed by multimodal imaging. Although these differences may influence procedural planning, pre-procedural adverse outcomes did not differ between sexes. Larger studies may help to better evaluate sex-related differences.

	Total (n=240)	Group A (n=121)	Group B (n=119)	p-value
Age at time of exam (yrs) – mean (SD)	81.72 (5.26)	82.44 (5.52)	82.62 (4.32)	p=0.763[b]
Follow-up period (months) - mean (SD)	9.85 (8.19)	9.50 (7.89)	10.21 (8.50)	p=0.623[b]
Medical history – no (%)				
Arterial hypertension	203 (84.6)	102 (84.3)	101 (84.9)	p=0.902[c]
Diabetes *mellitus*	81 (33.9)	42 (34.7)	39 (33.1)	p=0.786[c]
Dyslipidemia	156 (65.3)	79 (65.3)	77 (65.3)	p=0.995[c]
Overweight/Obesity	174 (73.1)	90 (75.0)	84 (71.2)	p=0.507[c]
Smoking (current or previous)	20 (8.4)	19 (15.7)	1 (0.8)	**p<0.001[c]**
Chronic kidney disease	21 (8.8)	13 (10.7)	8 (6.8)	p=0.279[c]
Atrial fibrillation/atrial flutter	65 (27.1)	37 (30.6)	28 (23.5)	p=0.219[c]
Heart Failure	24 (10.0)	13 (10.7)	11 (9.2)	p=0.699[c]
Myocardial infarction	14 (5.8)	9 (7.4)	5 (4.2)	p=0.285[c]
Prior percutaneous coronary intervention	23 (9.6)	17 (14.0)	6 (5.0)	**p=0.018[c]**
History of cancer	27 (11.3)	16(13.2)	11 (9.2)	p=0.329[c]
Cerebrovascular disease	32 (13.3)	16 (13.2)	16 (13.4)	p=0.960[c]
Medication – no (%)				
Aspirin and/or P2Y12 inhibitor and/or anticoagulation	150 (62.5)	83 (68.6)	67 (56.3)	**p=0.049[c]**
Betablocker	90 (37.7)	47 (38.8)	43 (36.4)	p=0.702[c]
Antiarrhythmics	15 (6.3)	9 (7.4)	6 (5.0)	p=0.443[c]
ARB/ARNI/ACEI	165 (69.3)	91 (75.2)	74 (63.2)	**p=0.045[c]**
Oral antidiabetics	79 (33.1)	44 (36.4)	35 (29.7)	p=0.271[c]
Diuretics	108 (45.0)	56 (46.3)	52 (43.7)	p=0.668[c]
Statins	170 (71.1)	84 (69.4)	86 (72.9)	p=0.555[c]
Symptoms– no (%)				
Fatigue	192 (81.0)	96 (80.0)	96 (82.1)	p=0.687[c]
Exertional angina	35 (14.8)	14 (11.7)	21 (17.9)	p=0.173[c]
Syncope	15 (6.3)	9 (7.5)	6 (5.1)	p=0.453[c]
CCT parameters				
Left ventricular ejection fraction - mean (SD)	62.3 (11.6)	60.2 (12.5)	67.4 (8.6)	**p=0.017[a]**
Mitral annulus calcification – no (%)	45 (18.8)	14 (11.6)	31 (26.1)	**p=0.004[c]**
Aortic valve calcium score - mean (SD)	2532 (1320.9)	3246 (1342.2)	1907 (934.8)	**p<0.001[a]**
TTE parameters - mean (SD)				
Left ventricular ejection fraction	58.8 (10.2)	58.0 (12.5)	62.3 (7.53)	**p<0.001[b]**
Septal thickness (mm)	14.5 (2.2)	15.6 (2.7)	14.9 (1.8)	**p=0.007[b]**
Posterior wall thickness (mm)	12.9 (1.74)	13.3 (1.59)	12.9 (1.65)	**p=0.010[b]**
E/A ratio	0.92 (0.46)	1.06 (0.59)	1.02 (0.55)	p=0.697[b]
E/e´	15.7 (5.6)	14.8 (5.2)	17.2 (6.0)	p=0.125[b]
Aortic valve peak velocity (m/s)	4.3 (0.56)	4.3 (0.61)	4.57 (0.51)	p=0.416[b]
Aortic valve mean gradient (mmHg)	46.4 (12.1)	45.7 (12.3)	52.5 (13.9)	p=0.145[a]
Aortic valve area (cm^2)	0.75 (0.16)	0.80 (0.14)	0.71 (0.17)	**p<0.001[a]**
Tricuspid Annular Plane Systolic Excursion	22.1 (4.0)	22.6 (3.7)	22.2 (3.2)	p=0.614[a]
Estimated pulmonary systolic arterial pressure	40.4 (12.3)	36.15 (8.6)	39.0 (9.8)	p=0.595[b]
Coronary angiography diagnosis– no (%)				
Obstructive coronary artery disease	74 (31.6)	50 (42.4)	24 (20.7)	**p<0.001[c]**
Pre-intervention outcomes – no (%)	**70 (29.5)**	**39 (32.5)**	**31 (26.5)**	**p=0.311[c]**
Cardiovascular hospitalization	62 (26.2)	36 (30.0)	26 (22.2)	p=0.173[c]
Heart failure hospitalization	40 (16.9)	23 (19.2)	17 (14.5)	p=0.341[c]
MACE	13 (5.5)	7 (5.8)	6 (5.1)	p=0.812[c]
Non-fatal acute myocardial infarction	4 (1.7)	3 (2.5)	1 (0.9)	p=0.622[d]
Non-fatal Stroke	3 (1.3)	1 (0.8)	2 (1.7)	p=0.619[d]
Cardiovascular mortality	9 (3.8)	4 (3.3)	5 (4.3)	p=0.747[d]
All-cause mortality	17 (7.2)	8 (6.7)	9 (7.7)	p=0.760[c]

Table 1. Demographic and clinical characteristics, imaging parameters and pre-procedures outcomes of patients undergoing transcatheter aortic valve implantation.

Statistical analysis: [a]T-student test, [b]Mann-Whitney U test, [c]Chi-square test, [d]Fisher´s exact test.

Abbreviations: ACEI - Angiotensin-Converting Enzyme Inhibitor; ARB - Angiotensin II Receptor Blocker; ARNI - Angiotensin Receptor-Neprilysin Inhibitor; CCT – Cardiac Computed Tomography; MACE - major adverse cardiovascular events; SD - Standard Deviation; TTE - Transthoracic Echocardiography.

Prevention Today, Sustainability Tomorrow: The Role of Specialist Community and Public Health Nurses in Cardiovascular Health

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Introduction

Cardiovascular diseases (CVDs) remain a leading cause of mortality, disability and healthcare expenditure worldwide and continue to represent a major public health challenge in Portugal. As most cardiovascular risk factors are modifiable, prevention, health literacy and community empowerment are key components of effective cardiovascular health strategies. Specialist Community and Public Health Nurses (SCPHNs) are well positioned to promote healthy lifestyles, support self-management and reduce cardiovascular risk at population level.

Objectives

To analyse the contribution of SCPHNs to cardiovascular disease prevention through health promotion, health literacy enhancement and community empowerment, highlighting their potential impact on National Health Service (NHS) sustainability.

Material and Methods

A narrative review of national and international scientific evidence, epidemiological reports and public health policy documents addressing cardiovascular disease prevention, stroke prevention, health literacy and community nursing interventions was conducted. The evidence was synthesised to identify the main contributions of SCPHNs to cardiovascular health outcomes and healthcare sustainability.

Results

Evidence indicates that SCPHN-led community interventions improve the control of cardiovascular risk factors, including hypertension, dyslipidaemia, diabetes, obesity and smoking. Health literacy initiatives are associated with improved treatment adherence, healthier lifestyles and increased participation in preventive programmes. Community nursing interventions also support early identification of cardiovascular risk, promote self-management and strengthen community engagement. These outcomes contribute to reductions in hospitalisations, cardiovascular events and premature mortality, particularly stroke-related mortality, while promoting more efficient use of healthcare resources. Furthermore, prevention-focused strategies have demonstrated favourable cost-effectiveness, supporting long-term NHS sustainability.

Conclusions

Cardiovascular prevention should be considered a strategic priority for health systems facing increasing demographic and epidemiological challenges. SCPHNs play a pivotal role in improving health literacy, empowering communities and implementing evidence-based preventive interventions. Strengthening community-based cardiovascular prevention programmes may reduce disease burden, improve quality of life and contribute to a more sustainable and resilient NHS. Investing in prevention today represents an opportunity to achieve better population health outcomes while optimising healthcare resources for future generations.

FROM TREATMENT TO PREVENTION:
EMPOWERING COMMUNITIES FOR CARDIOVASCULAR HEALTH AND NATIONAL HEALTH SERVICE SUSTAINABILITY

The role of specialist community and public health nurses

(R)Evolução Cardiovascular,
Impacto clínico e desafios para o SNS
16th CHALLENGES in CARDIOLOGY
3 July 2026 | Hotel Vila Baleeira

1. INTRODUCTION

Cardiovascular diseases (CVD) remain one of the leading causes of mortality, disability and healthcare expenditure worldwide and continue to represent a major public health challenge in Portugal.

Most cardiovascular risk factors are modifiable, highlighting the importance of prevention, health literacy and community empowerment.

Specialist community and public health nurses are strategically positioned to promote healthy lifestyles and reduce cardiovascular risk at population level.

CARDIOVASCULAR DISEASE IN PORTUGAL LATEST DATA (2024)

119 046
deaths in Portugal (2024)

MAIN CAUSES OF DEATH
(Diseases of the circulatory system remain among the leading causes)

9 007 DEATHS DUE TO STROKE (7.6%)

6 470 DEATHS DUE TO ISCHAEMIC HEART DISEASE (5.5%)

RELEVANCE FOR THE NATIONAL HEALTH SERVICE

- CVD are responsible for a substantial proportion of healthcare costs: hospitalisations, consultations, diagnostic exams and long-term medication.
- High indirect costs due to disability, loss of productivity and early mortality.
- Prevention and health promotion are cost-effective strategies that contribute to a more sustainable National Health Service (NHS).

MODIFIABLE CARDIOVASCULAR RISK FACTORS

- Arterial hypertension
- Smoking
- Unhealthy diet
- Physical inactivity
- Obesity
- Diabetes mellitus

Up to 80% of premature cardiovascular events can be prevented by addressing these risk factors.

Source: DGS, 2025; WHO, 2024.

2. OBJECTIVES

To analyse the contribution of specialist community and public health nurses to cardiovascular disease prevention through health promotion, health literacy enhancement and community empowerment, highlighting their potential impact on NHS sustainability.

3. MATERIAL AND METHODS

Narrative review and critical analysis of national and international scientific evidence, epidemiological reports and public health policy documents related to cardiovascular disease prevention, stroke prevention, health literacy and community nursing interventions.

4. THE ROLE OF SPECIALIST COMMUNITY AND PUBLIC HEALTH NURSES

Health education and health literacy improvement
Simplifying information, increasing knowledge and supporting informed decision-making.

Cardiovascular risk assessment and early identification
Screening, risk stratification and referral in the community.

Promotion of healthy lifestyles
Encouraging healthy eating, physical activity, weight control and smoking cessation.

Therapeutic adherence and self-management support
Empowering people to manage their condition and adhere to treatment.

Community empowerment and participation
Developing skills, building confidence and promoting community involvement.

HEALTH LITERACY MATTERS

Low health literacy is associated with:

- poorer health behaviours
- lower adherence to treatment
- higher use of emergency services
- worse health outcomes
- higher healthcare costs

of Portuguese adults have inadequate or problematic health literacy

(Arriaga et al., 2022)

Improving health literacy enables people to make better choices, adopt healthier lifestyles and participate actively in their health.

5. RESULTS – WHAT DOES THE EVIDENCE SAY?

Evidence shows that community-based interventions led by nurses and focused on prevention and health literacy contribute to:

- Better control of cardiovascular risk factors (hypertension, cholesterol, diabetes, obesity, smoking)
- Greater adherence to medication and follow-up appointments
- Increased physical activity and healthier diets
- Reduction of hospitalisations for cardiovascular events and complications
- Reduction of premature mortality, including stroke mortality

PREVENTING STROKE: A PRIORITY

Stroke remains:

- One of the leading causes of death in Portugal (9 007 deaths in 2024):
- A major cause of disability and loss of independence.

ACT FAST – SAVE LIVES

F	A	S	T
FACE Drooping?	ARMS Weakness?	SPEECH Difficulty?	TIME Call 112 immediately!

6. IMPACT ON THE NHS

- Preventing cardiovascular events reduces pressure on emergency services and hospital care.
- Promotes more efficient use of resources and reduces long-term costs.
- Contributes to a healthier population, with more years of life in good health.
- Supports the sustainability and resilience of the National Health Service.

7. CONCLUSIONS

Cardiovascular prevention must be a strategic priority for the sustainability of the NHS. Specialist community and public health nurses play a crucial role in improving health literacy, empowering communities and implementing evidence-based preventive interventions.
Investing in prevention today means fewer cardiovascular events tomorrow, better quality of life and a more sustainable health system.

RECOMMENDATIONS

- Strengthen community-based cardiovascular prevention programmes.
- Invest in health literacy, tailored communication and population empowerment.
- Promote intersectoral collaboration for healthy environments and active communities.
- Value and integrate specialist community and public health nurses as key agents in prevention.

CONCEPTUAL MODEL

From literacy to sustainability

Empowered people · Healthier communities · Sustainable future

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PREVENT TODAY. EMPOWER ALWAYS. SAVE TOMORROW.

COMMUNITY NURSING · HEALTH LITERACY · PREVENTION · SUSTAINABILITY

HIV-1 genetic variability, antiretroviral resistance, and cardiovascular diagnoses in a Brazilian deterministic linkage cohort

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Introduction

People living with HIV have increased cardiovascular risk, but population-based research is limited by disconnected data systems. Deterministic linkage using composite keys can integrate morbidity, mortality, laboratory, and genotypic records, enabling the study of underexplored cardiovascular determinants, including HIV-1 subtype and antiretroviral resistance mutations.

Objectives

To develop a national deterministic linkage cohort of people living with HIV in Brazil and assess associations between HIV-1 subtype, resistance mutations, biological sex, and cardiovascular diagnoses.

Material and methods

We conducted a retrospective observational study using Brazilian national databases. Hospital Information System, Mortality Information System, and SISGENO/SISCEL laboratory/genotypic records were integrated. The workflow included variable standardization, date harmonization, deduplication, temporal auditing, and deterministic linkage through composite keys using Python and DuckDB. HIV-1 subtypes and resistance mutations were identified from pol sequencing. Cardiovascular diagnoses were defined using ICD-10 codes. Sex-stratified multivariable logistic regression estimated adjusted odds ratios controlling for age and cardiovascular risk factors.

Results

The SIH-SIM linkage consolidated 736,783 eligible records. The final biologically enriched cohort included 20,692 individuals receiving antiretroviral therapy; 55.0% were men, and mean age was 38.4±12.0 years. Median viral load was 22,640 copies/mL, 39.5% had CD4+ T-cell count <200 cells/mm³, and subtype B was most frequent. Cardiovascular diagnoses were identified in 1,022 individuals (4.79%); mean age was 43.1±10.0 years. Frequent diagnoses included acute myocardial infarction, essential hypertension, pulmonary embolism, and heart failure. Compared with subtype B, subtype C was associated with lower cardiovascular odds in men (OR 0.65; p=0.001) and women (OR 0.61; p<0.001). Resistance mutations showed a sex-specific pattern. In women, M41L (OR 1.72; 95% CI 1.33-2.23; p=0.0001), M46I (OR 1.51; 95% CI 1.10-2.08; p=0.0142), T215F (OR 1.51; 95% CI 1.07-2.12; p=0.0235), and L33F (OR 1.53; 95% CI 1.07-2.18; p=0.0236) were associated with higher cardiovascular odds. In men, no consistent pattern of increased odds was observed; L33F showed an isolated inverse association (OR 0.65; 95% CI 0.44-0.95; p=0.0223).

Conclusions

Deterministic linkage enabled an individualized, biologically enriched national cohort. HIV-1 subtype, antiretroviral resistance, and biological sex were associated with cardiovascular diagnoses, supporting viral biomarkers for sex-sensitive cardiovascular risk stratification in people living with HIV