



13th **CHALLENGES in** **CARDIOLOGY**

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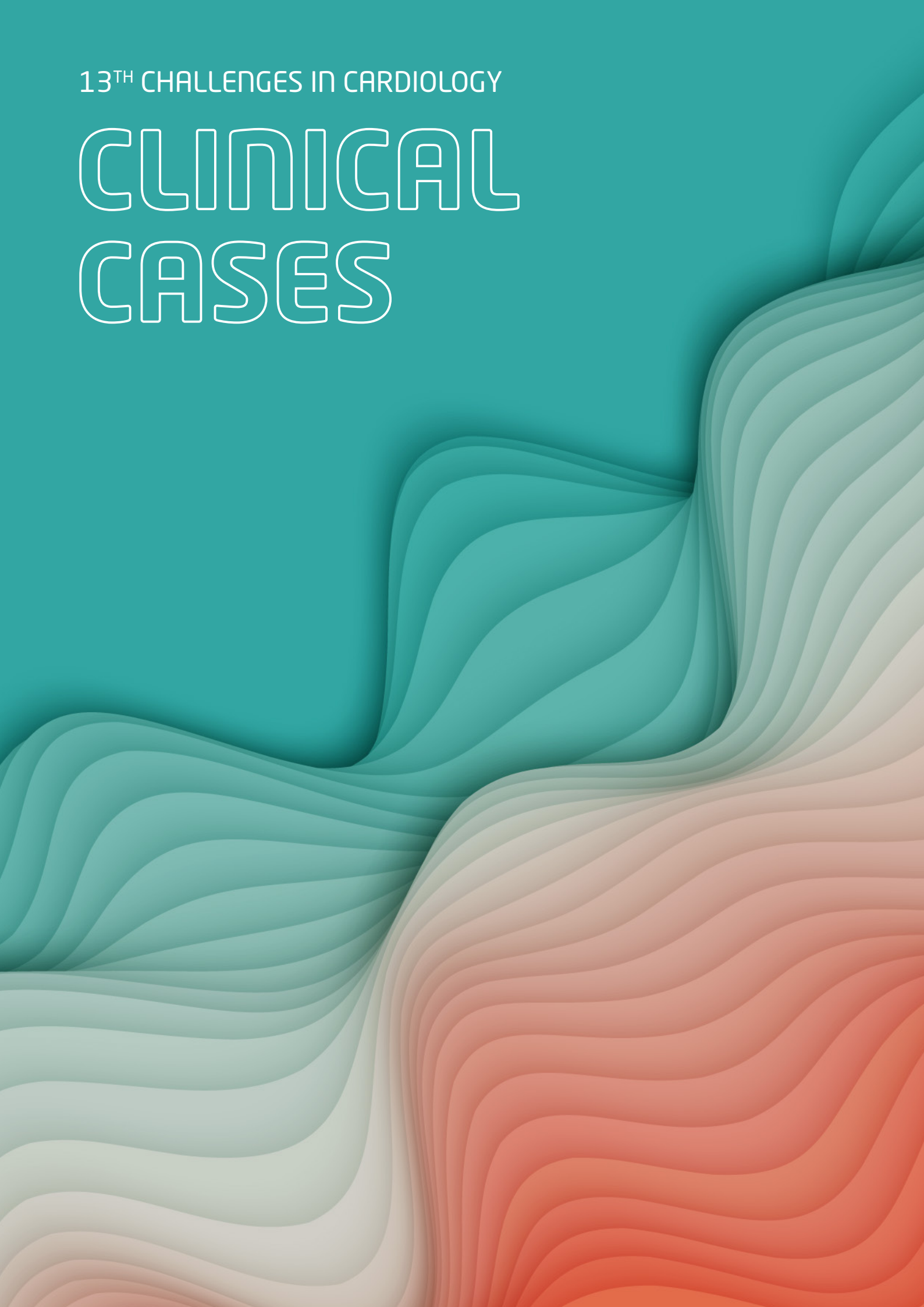
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13TH CHALLENGES IN CARDIOLOGY

CLINICAL CASES



CC 06

Infective endocarditis post-transcatheter aortic valve implantation: an entity to be aware of

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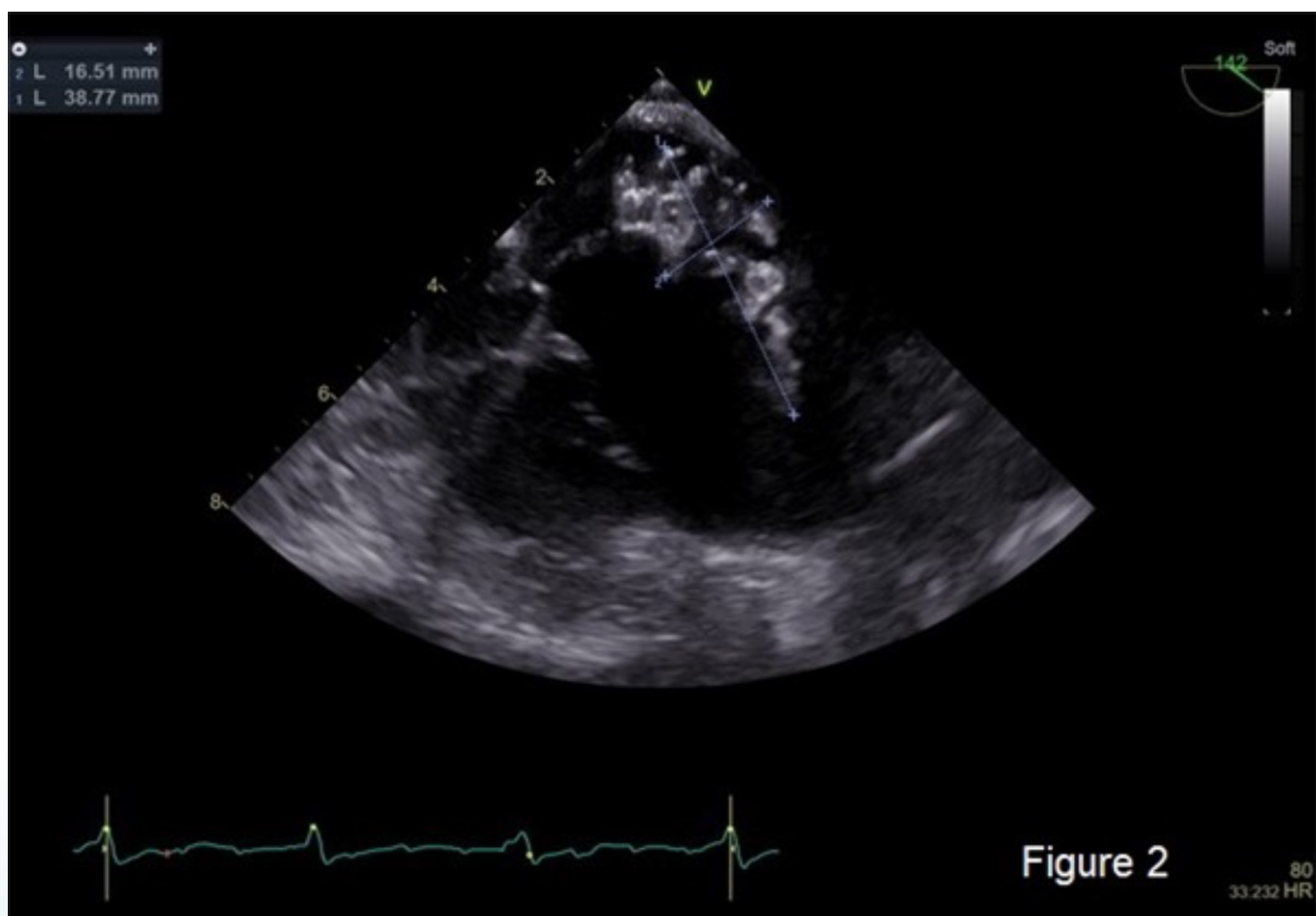
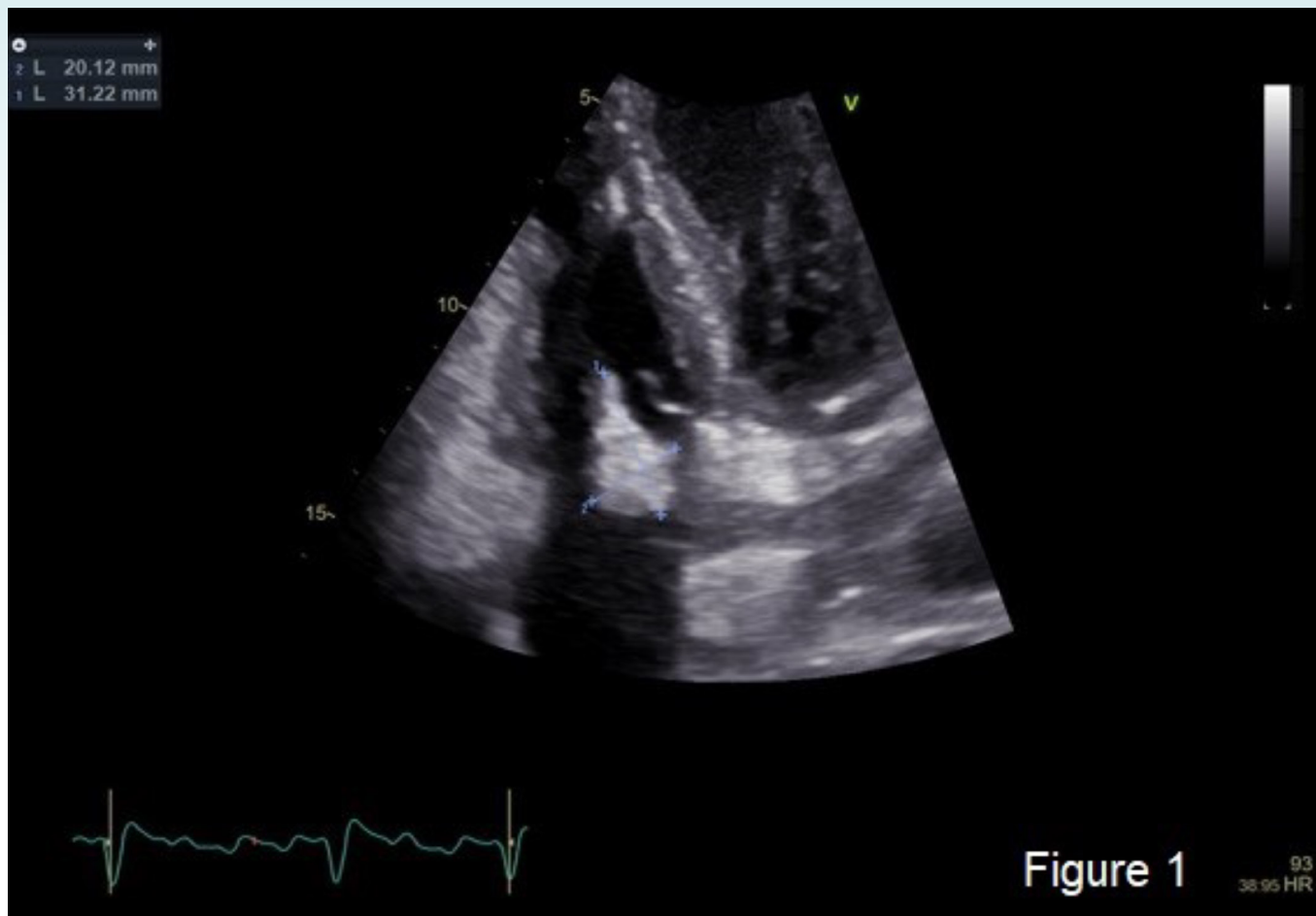
An 85-year-old man, with a known history of myelodysplastic neoplasm, paroxysmal atrial fibrillation, arterial hypertension and dyslipidemia, underwent a transcatheter aortic valve percutaneous (TAVI) ACURATE neo2™ implantation due to symptomatic severe aortic stenosis. Pacemaker implantation was needed owing to a complete auriculoventricular block after the procedure.

Two months later, he presented to the Emergency Department referred by his cardiologist for evidence of an intracardiac mass on a transthoracic echocardiogram (TTE). On admission, he was hemodynamically stable, with no signs of congestion, but febrile. He presented thinness, frailty and a lowered level of consciousness. A cranial computed tomography revealed no acute lesions, but signs of cortical atrophy. Blood tests showed elevated inflammatory parameters, anemia and acute kidney injury. Blood cultures were collected and empirical antibiotic therapy was started.

The TTE revealed the presence of a mobile mass attached to the ventricular lead in the right chambers (Figure 1), with 33 mm of diameter and high embolic potential, associated with mild tricuspid regurgitation. The biological prosthesis had two *de novo* paravalvular leaks, a mild anterior one and a moderate posterior one.

Based on the presence of one major criterion and one minor criterion for infective endocarditis (IE), the diagnosis of possible IE was established. Antibiotic therapy was changed to vancomycin, rifampicin and ceftriaxone. Medical treatment was decided due to the patient's frailty and lack of embolization or severe valvular dysfunction. Blood cultures were negative. After eight days of treatment, inflammatory parameters decreased, but renal and neurological function deteriorated. Transesophageal echocardiography revealed mass disruption, with a 38mm diameter (Figure 2) and evidence of a second, smaller, mass in the auricular lead. He deteriorated clinically and analytically and ended up dying.

In conclusion, the authors present a case of post-TAVI infective endocarditis affecting the pacemaker leads and possibly the bioprosthesis. Its incidence is low, but it tends to grow in parallel with the increase in TAVI implantation. Morbimortality is not negligible, in part due to the advanced age and comorbidities of these patients, so physicians must be aware of this entity to act promptly.



CC 08

Late Right Atrial Rooftop Perforation Associated to Atrial Septal Defect Closure Device induced erosion

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Atrial septal defect (ASD) is the second most common congenital heart defect in adults. Percutaneous closure using metal devices is a widely accepted alternative to surgical repair, with at least similar results and reduced rate of early complications and length of stay.¹ Although infrequent, potentially severe late complications may occur and require a high level of suspicion in order to reach a diagnosis.

A 48-year-old woman admitted to the Emergency Department (ED) with complaints of chest pain, malaise and nausea. She had previous history of atrial septal defect (ASD) closure performed percutaneously 11 years prior (Occlutech Figulla ASD Occluder). Vital signs were normal and laboratory tests were unremarkable.

Electrocardiogram showed sinus tachycardia. Thoracoabdominopelvic computerized tomography revealed a moderate pericardial effusion. No signs of pulmonary embolism or acute aortic dissection were seen.

During the surveillance period in the ED, the patient became hemodynamically unstable. Point of care ultrasonography revealed pericardial effusion with cardiac tamponade. An emergent subcostal pericardiocentesis was performed and a small amount of hematic fluid drained, resulting in clinical stabilization. The patient was admitted for further study.

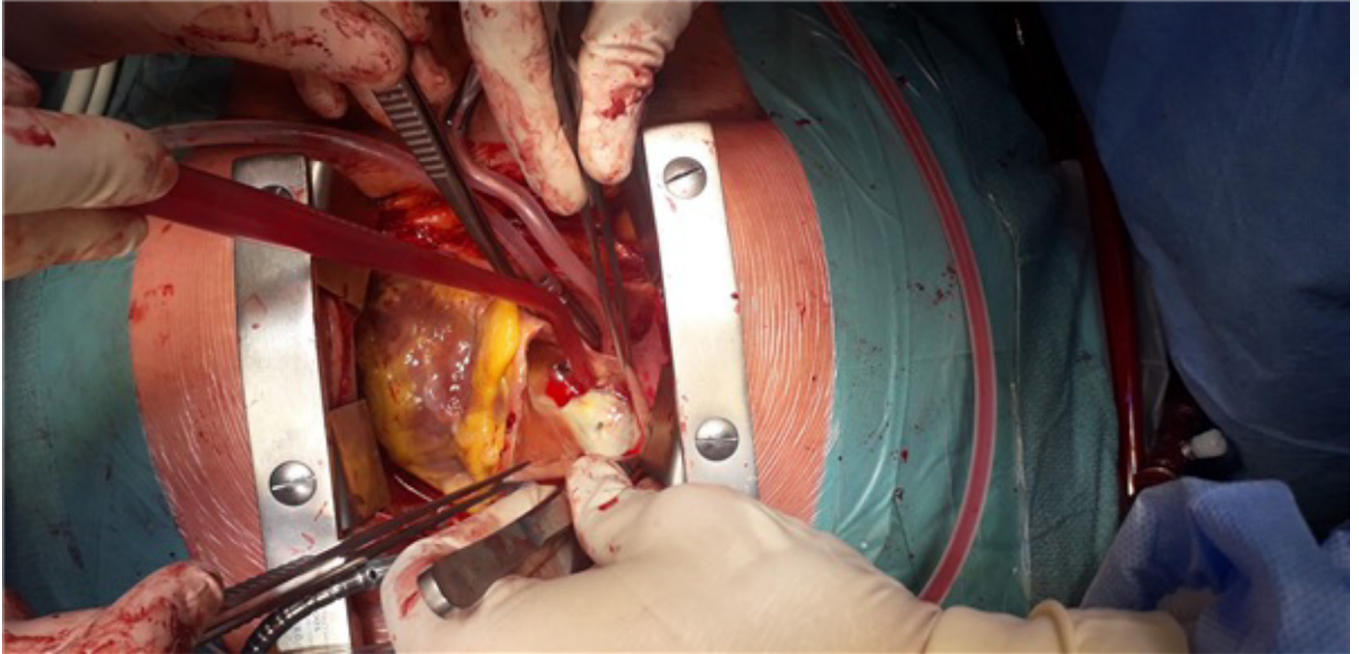
Formal transthoracic echocardiogram showed a left ventricle of normal dimensions with normal ventricular systolic function. Normal valvular structures. Interatrial clo-

sure device appeared to function normally, and no leaks were seen. A minimal pericardial effusion remained. The biochemical study of the pericardial fluid was compatible with hemopericardium.

Transesophageal echocardiogram showed, at the 0°/180° plane, the edge of the device impinging the roof of the atria, with a nearby hypoechogenic pericardial effusion (maximum 3 mm). Ultrasonic contrast injection was tried, without any leakage to the pericardial space.

The patient remained stable for the following days. However, the exact cause of the hemopericardium was yet to be determined. After multidisciplinary discussion with Cardiothoracic Surgery, it was decided to perform elective surgical exploration. The surgery revealed a normal sized heart, surrounded by a small amount of hematic fluid, and a slight protrusion of the device through the right atrial roof, where an already healed thickening could be found. Right atriotomy revealed protrusion of the right disc over the aortic torus and roof of the right auricle. The device was explanted, and the erosion site was repaired, as well as the ASD. Post-operative period was uneventful.

Late ASD closure device erosion is rare and can occur many years after the percutaneous procedure.¹⁻² We report the case of a patient who presented with cardiac tamponade due to late device-induced erosion of the atrial roof. A high index of suspicion is crucial to timely identify and manage this potentially fatal complication.¹⁻²



CC 09

Bubblegum Heart

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53 year old male with multiple CV risk factor and stage 3 CKD. Resorted to the ED with complaints of progressively dyspnea, cough without sputum, wheezing, fever (41°C), pleuritic chest pain. He presented with signs of respiratory distress and fever (38.0°C).

ECG presented sinus tachycardia, T wave inversion/flattening at V5 - V6. He had anaemia, elevated inflammatory parameters, abnormal renal function and elevated MNM. Contrast chest CT revealed left sided pleural effusion, small pericardial effusion, and no signs of pulmonary thromboembolism or acute aortic syndrome. TTE on admission demonstrated depressed LV systolic function with apparent basal lateral pseudoaneurysm and severe mitral valve regurgitation.

The patient was admitted to the C-ICU and started on empirical antibiotic therapy (amoxicillin / clavulanic acid and clarithromycin) for pneumonia.

TEE revealed a mass on the posterior leaflet of the mitral valve, mobile, with important prolapse of this leaflet, and severe mitral regurgitation.

A new diagnosis was considered - possible native mitral valve infective endocarditis complicated with perivalvular abscess and pseudoaneurysm.

CMR demonstrated left heart chamber dilation with mildly depressed of global systolic function, marked increase in the thickness of the basal segment of the anterolateral wall and probable pseudoaneurysm of the basal part of the anterolateral wall. During this exam the patient presented with severe desaturation and clinical instability, forcing the premature interruption of the exam.

The patients clinical condition worsened over the next few days, He was transferred to Cardiothoracic Surgery. Due to personal religious beliefs, he refused blood transfusions. After a deliberation period, the patient maintained his decision and was therefore refused to surgery.

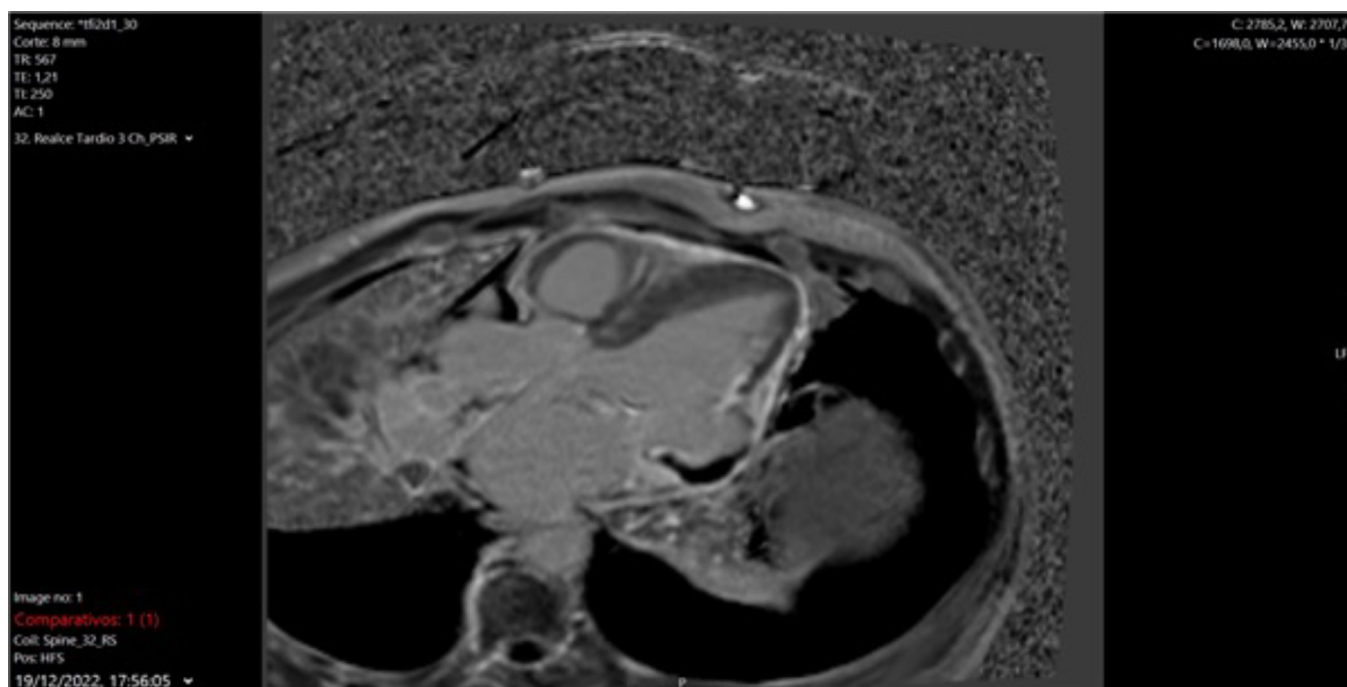
He progressively stabilized, his kidney function improved slightly and hemoglobin levels remained stable.

Reevaluation TTE revealed on the basolateral segment, an image suggestive of pseudoaneurysm / contained rupture.

New CMR showed a pseudoaneurysm of the left ventricle (51 x 25 mm) originating from a continuity solution of the basal segment of the anterolateral wall with thrombotic deposits inside.

Our diagnostic hypothesis was again reconsidered, assuming as most likely diagnosis subacute MI of the basal anterolateral wall complicated with pseudoaneurysm and mitral cord rupture with severe mitral regurgitation.

Medical therapy was then our only option at the time. He was kept on single antiplatelet therapy and high-potency statin and completed over 40 days of antibiotics (ceftriaxone and ampicillin). Kidney function progressively improved. Endoscopy studies were normal and with iron supplementation his hemoglobin levels improved. He presented gradual clinical improvement and was asymptomatic from a cardiopulmonary standpoint at the time of discharge.



CC 10

Recurrent myocarditis: the defiant way until definitive etiological diagnosis

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INTRODUCTION

Myocarditis is an inflammatory disease of the myocardium with a challenging diagnosis due to heterogeneity of clinical presentation and an incidence difficult to determine since endomyocardial biopsy (EMB), the diagnostic gold standard, is used infrequently. Although its etiology often remains undetermined, viral infection is the most important cause of myocarditis in Europe.

CASE REPORT

In January 2020, a 19-year-old woman is admitted in the emergency department due to rest chest pain exacerbated when breathing deeply, with a 4-hour duration. Serial electrocardiogram showed sinus rhythm with no dynamic ST segment alterations. High sensitivity troponin (hs-troponin) was 4282pg/mL with negative inflammatory parameters. Transthoracic echocardiography revealed preserved biventricular systolic function with normal segmental contractility and valve function and no pericardial effusion. Coronary angiogram excluded coronary artery anomalies and disease. A probable acute myocarditis was assumed. She evolved favorably with no heart failure signs and no dysrhythmic events. Ambulatory cardiac magnetic resonance imaging (MRI) later revealed inflammation and fibrosis in the subepicardial lateral wall consistent with the clinical diagnosis.

In July and December 2020, January 2021 and March, August and October 2022, she had recurrent episodes of acute myocarditis, with similar clinical presentation and hs-troponin elevation. Seriated cardiac MRI revealed

progressive areas of fibrosis consistent with the diagnosis. Immunological study was positive for antinuclear antibodies (ANA) and smooth-muscle antibodies both in a titer of 1:320. Infectious serological analyses were irrelevant. After the fourth episode, it was decided in a multidisciplinary team to initiate immunosuppressive therapy in the suspicion of an auto-immune etiology. After a longer period without events, acute myocarditis recurred. EMB performed in October 2022 showed interstitial fibrosis compatible with a previous myocardial injury, with no evidence of inflammation. Cardiotropic viruses assay was negative.

She is currently awaiting arrhythmogenic left ventricular cardiomyopathy (ALVC) genetic test results, with no recurrence of events in a 7-month period.

DISCUSSION

This case report demonstrates the need for a high level of suspicion in making the diagnosis of myocarditis, as EMB is not widely available and has low sensitivity. As in this case, cardiac MRI is an alternative diagnostic modality, with the additional advantage of being noninvasive.

The way until etiological diagnosis is also defiant, persisting undetermined in some cases. Since EMB showed no signs of auto-immune inflammation, the benefit of keeping immunosuppressive therapy should be evaluated. In this patient, considering the fibrosis extent, it is also mandatory to exclude ALVC.

CC 11

The value of modified multimodality cardiac imaging

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INTRODUCTION

Ventricular tachycardia is a potentially life-threatening arrhythmia, and it is responsible for a significant percentage of sudden cardiac deaths. The exact cause of ventricular tachycardia isn't always known, and it could be the first manifestation of an underlying disease, triggered by another heart condition and in certain forms of ventricular tachycardia it represents an inherited disease.

CASE SUMMARY

We report a case of a 60-year-old female, with previous history of hypothyroidism, chronic venous insufficiency and psoriatic arthritis; and known cardiovascular risk factors: hypertension, dyslipidemia, and obesity. There was no personal or family history of unexplained syncope or cardiac arrest.

She was admitted to the Emergency Department complaining of sudden chest pain and palpitations while working. On initial evaluation, she was conscious, tachycardic, hypotensive and the EKG revealed a ventricular tachycardia at 220 bpm. The patient recovered sinus rhythm after a synchronized electric cardioversion with 100J.

Laboratory analysis excluded significant electrolyte abnormalities and a borderline elevation in Troponin T (cTnT-us 46 ng/L) and NT-proBNP levels (NTproBNP 137 pg/mL).

A bedside TTE revealed a normal left ventricle (LV) with a preserved LVEF, and a right ventricular outflow tract (RVOT) dilatation with a free wall dyskinesia and a sub valvular aneurysm.

Cardiac CT study excluded significant coronary artery disease, although morphological changes of the right ventricle (RV) with RVOT aneurysms were noted (Figure 3).

Cardiac magnetic resonance imaging showed a preserved ejection fraction with bright blood images in the right ventricular outflow tract (RVOT) plane and microaneurysms in the RV free wall.

The diagnosis of Arrhythmogenic Right Ventricular Dysplasia (ARVD) was made, and the patient was discharged after a single-chamber implantable cardioverter defibrillator (ICD) being placed and on sotalol 80 mg twice daily.

DISCUSSION

ARVD is a primary cardiomyopathy most commonly diagnosed after an individual presents an arrhythmia. Commonly, diagnostic criteria rely on a combination of EKG, imaging studies and a documented arrhythmia (Marcus *et al*, 2010). Multimodality cardiac imaging with modified views/planes play an important role and has become a cornerstone for establishing the diagnosis in this case.

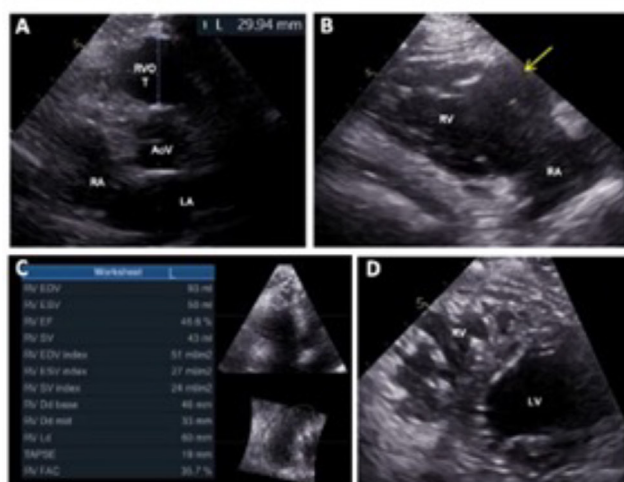


Fig. 1– Transthoracic echocardiogram study. A – RVOT measurement from the parasternal short-axis view; B – modified RV view with subvalvular aneurysm (yellow arrow); C – RV measurements; D – Trabeculation of RV in a parasternal short-axis view. AoV = aortic valve; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; RVOT = right ventricular outflow tract.

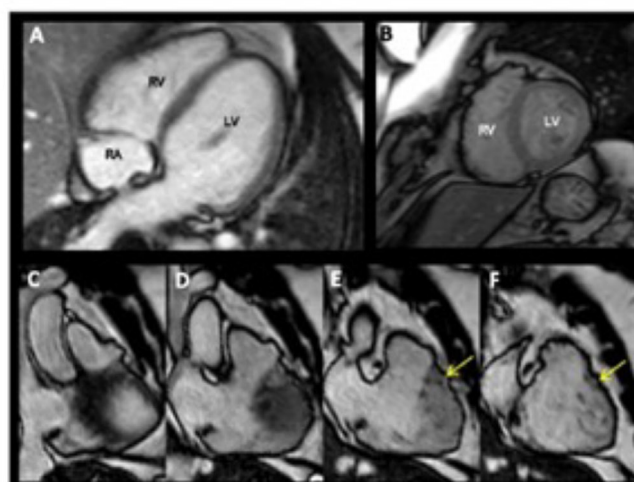


Fig. 2– CMR study showing conventional four and two-chamber LV views without significant changes (A-B). C-F: Additional CMR planes showing bright blood images in the right ventricular outflow tract (RVOT) plane and microaneurysms (yellow arrows) in the RV free wall. LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

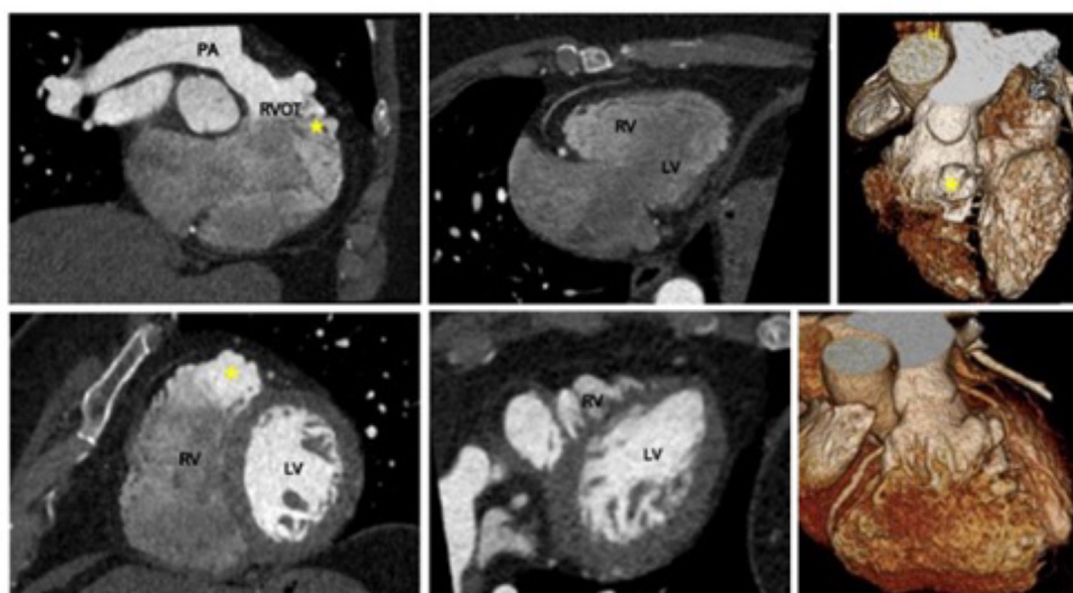


Fig.3– Cardiac CT study showing morphological changes of the right ventricle (RV) with right ventricular outflow tract aneurysms (RVOT) (★). PA– Pulmonary artery, LV – Left ventricle.

CC 14

A Rare Encounter: Cardiac Angioma in an Adult Patient

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INTRODUÇÃO

Cardiac masses encompass a wide spectrum of etiologies, and the differential diagnosis can be challenging. This clinical case intends to highlight the complexities in its diagnosis and management of these patients.

CASE PRESENTATION

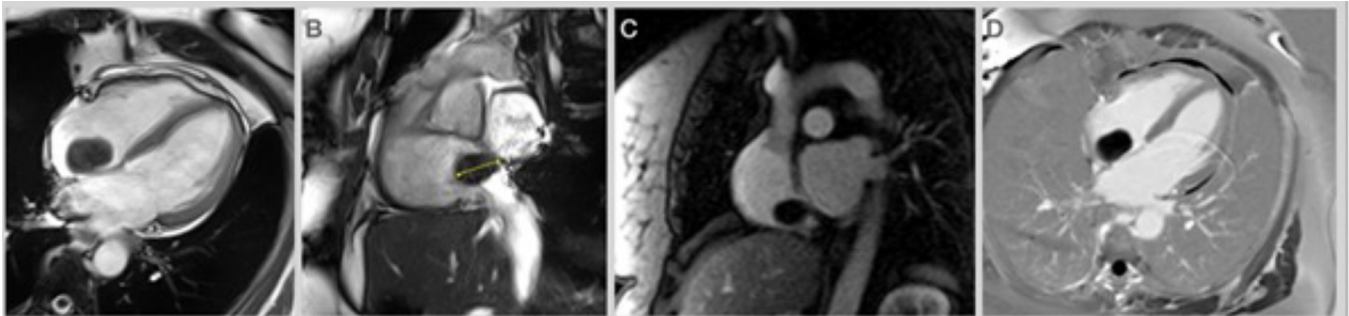
A 60-year-old woman was referred to a Cardiology Consultation as an outpatient due to complaints of oppressive chest pain unrelated to effort over the past months. She had no further complaints. Physical examination was unremarkable. Her electrocardiogram (ECG) showed sinus rhythm, with no further changes. A transthoracic echocardiogram (TTE; **Panel 1**) revealed in the right atricle, in possible relation to the Eustachian valve, a rounded, echogenic, mobile mass of doubtful etiology, with about 6 mm in diameter, was found. A transesophageal echocardiogram (TEE; **Panel 1**) showed, at the level of the outflow of the inferior vena cava, a voluminous rounded mass (25x32mm), pediculated, with well-defined borders and capsulated aspect (echogenic borders with apparently hypoechogenic solid interior in relation to the cardiac walls). Cardiac magnetic resonance imaging (CMR; **Panel 2**) disclosed in the right atricle, a well delimited, mobile mass, 28x15x25 mm in size, with apparent origin in the lower portion of the interatrial septum. This mass was isointense on T1, isointense on T2, without perfusion on first pass images and without early or late enhancement. These findings could be compatible with the diagnosis of myxoma, but a definitive diagnosis

could not be established. The patient was referred for surgical excision of the mass. Surgical resection was successful and the post-operative period was unremarkable. Histopathological analysis of the removed tissue allowed for the definitive diagnosis of cardiac angioma.

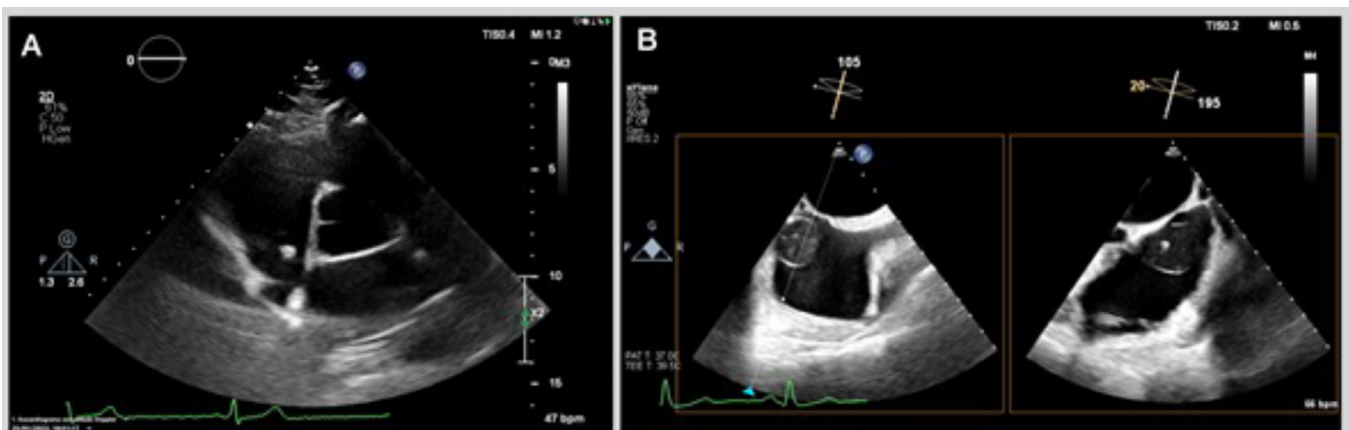
DISCUSSION

The differential diagnosis of cardiac masses presents significant challenges. Differentiating between benign and malignant masses, as well as identifying specific tumor types, is crucial for appropriate management and patient outcomes. While imaging techniques play a crucial role in the initial evaluation, they may not always provide definitive differentiation between benign and malignant lesions or specific tumor types. Histopathological analysis plays a pivotal role in achieving an accurate diagnosis, guiding treatment decisions, and optimizing patient care.

Cardiac angioma is a rare benign tumor that originates from blood vessels in the heart, with higher incidence in children and infants. Due to its rarity, there is limited literature available specifically focusing on adult cases. Patient management requires a multidisciplinary approach. Surgical resection is the primary treatment modality. They are typically considered benign tumors. Regular follow-up and surveillance are important after surgical resection, including periodic imaging evaluations to monitor for signs of recurrence or residual disease.



Panel 2: Cardiac magnetic resonance disclosed in the right auricle, a well delimited, mobile mass (A), 28 x 15 x 25 mm in size (B), with apparent origin in the lower portion of the interatrial septum. This mass was isointense on T1, isointense on T2, without perfusion on first pass images (C) and without early or late enhancement (D).



Panel 1: (A) transthoracic echocardiogram PSAX at the level of the aortic valve, showing in the right auricle, in possible relation to the Eustachian valve, a rounded, echogenic, mobile mass of doubtful etiology, with about 6 mm in diameter, was found; (B) transesophageal echocardiogram mid-esophageal bicaval view and X-plane, showing, at the level of the outflow of the inferior vena cava, a voluminous rounded mass (25x32mm), pediculated, with well-defined borders and capsulated aspect (echogenic borders with apparently hypoechogenic solid interior in relation to the cardiac walls).

CC 15

Apical thrombus in a patient with biventricular Takostubo

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INTRODUÇÃO

Takotsubo Syndrome is a challenging diagnosis characterized by temporary wall motion abnormalities that extend beyond the distribution of a single coronary artery territory. Although it most frequently involves the left ventricle, right ventricular involvement can also be present.

CASE PRESENTATION

An 82 year old woman, with type 2 diabetes and no other relevant past medical history, was brought to the emergency room after being found after unconscious in her home. She had no recollection of what happened and was last seen well the day before. Physical examination was unremarkable.

Electrocardiogram on showed sinus tachycardia, left anterior fascicular block, poor R wave progression in precordial leads and by-phasic T wave in V5 and inverted T wave in V6.

Initial blood work revealed leukocytosis with neutrophilia, elevated CRP, and elevated myocardial necrosis markers. Summary of urine with leukocytes and positive nitrites. Abdominopelvic CT showed signs of focal pyelonephritis, without local complications.

Summary transthoracic echocardiogram showed normal-sized cardiac cavities and moderate left ventricular dysfunction with apical ballooning.

Head CT revealed no acute lesions and head CT angiogram showed no great vessel occlusion. The patient was evaluated by Neurology and lumbar puncture was performed and excluded CNS infection.

She was admitted to the Intermediate Cardiac Care Unit with presumptive diagnosis of Takotsubo Syndrome, with urinary track infection as the assumed triggering factor. She presented a favorable evolution, with good clinical and analytical response to antibiotics. From a cardiac point of view, the patient also evolved favorably, with maintained clinical, electrical and hemodynamic stability.

Cardiac magnetic resonance showed findings compatible with the clinical suspicion of Takotsubo Syndrome with biventricular involvement, with moderate depression of Left ventricular function (ejection fraction of 38%) and the presence of apical thrombus.

The patient was hypocoagulated, initially with enoxaparin, which was later switched to warfarin. She was also medicated with ACE inhibitor and beta-blocker.

Echocardiographic reevaluation before discharge revealed partial improvement in left ventricular systolic function, with an ejection fraction of 42%, maintaining apical akinesia and hypokinesia of the distal segments of all ventricular walls and the interventricular septum.

CONCLUSION

Left ventricular thrombi during the acute phase of Takotsubo Syndrome occur in 2.2% of cases. Presence of apical ballooning and higher levels of troponin I on admission are associated with an increased risk. It can be successfully managed with 3 months of oral anticoagulation therapy with complete resolution. Long-term survival rates of patients with left ventricular thrombosis are similar to those of patients without.

CC 16

Prosthetic valve thrombosis: a challenging clinical case presenting with cardiogenic shock

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INTRODUCTION

Prosthetic valve thrombosis (PVT) is a potentially life-threatening complication of valve replacement, that can result in hemodynamically severe stenosis or regurgitation. Suspicion of PVT warrants rapid diagnosis evaluation, with echocardiography playing an important role. The treatment of choice will be influenced by clinical status, presence of valvular obstruction, valve location and thrombus size.

CASE REPORT

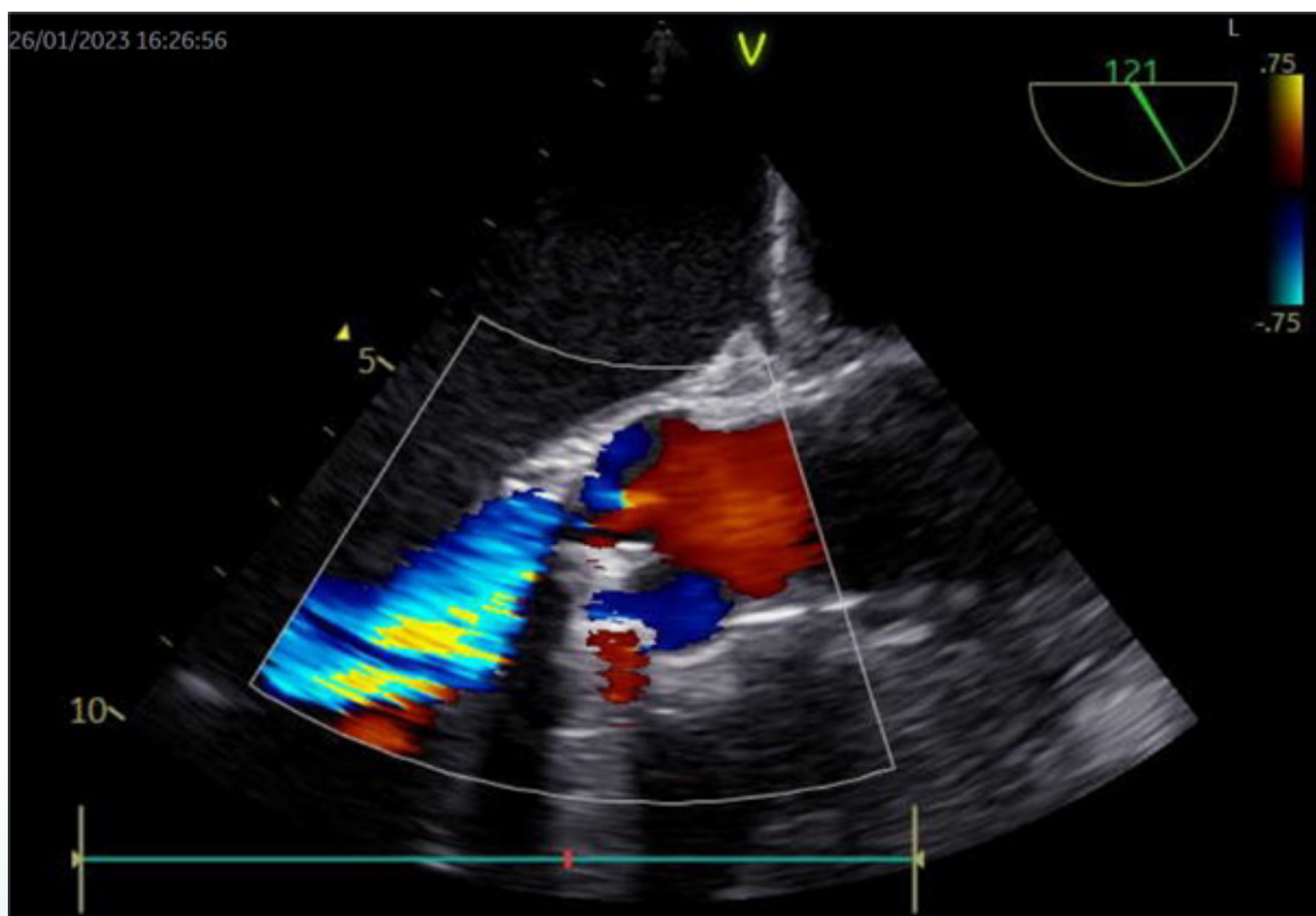
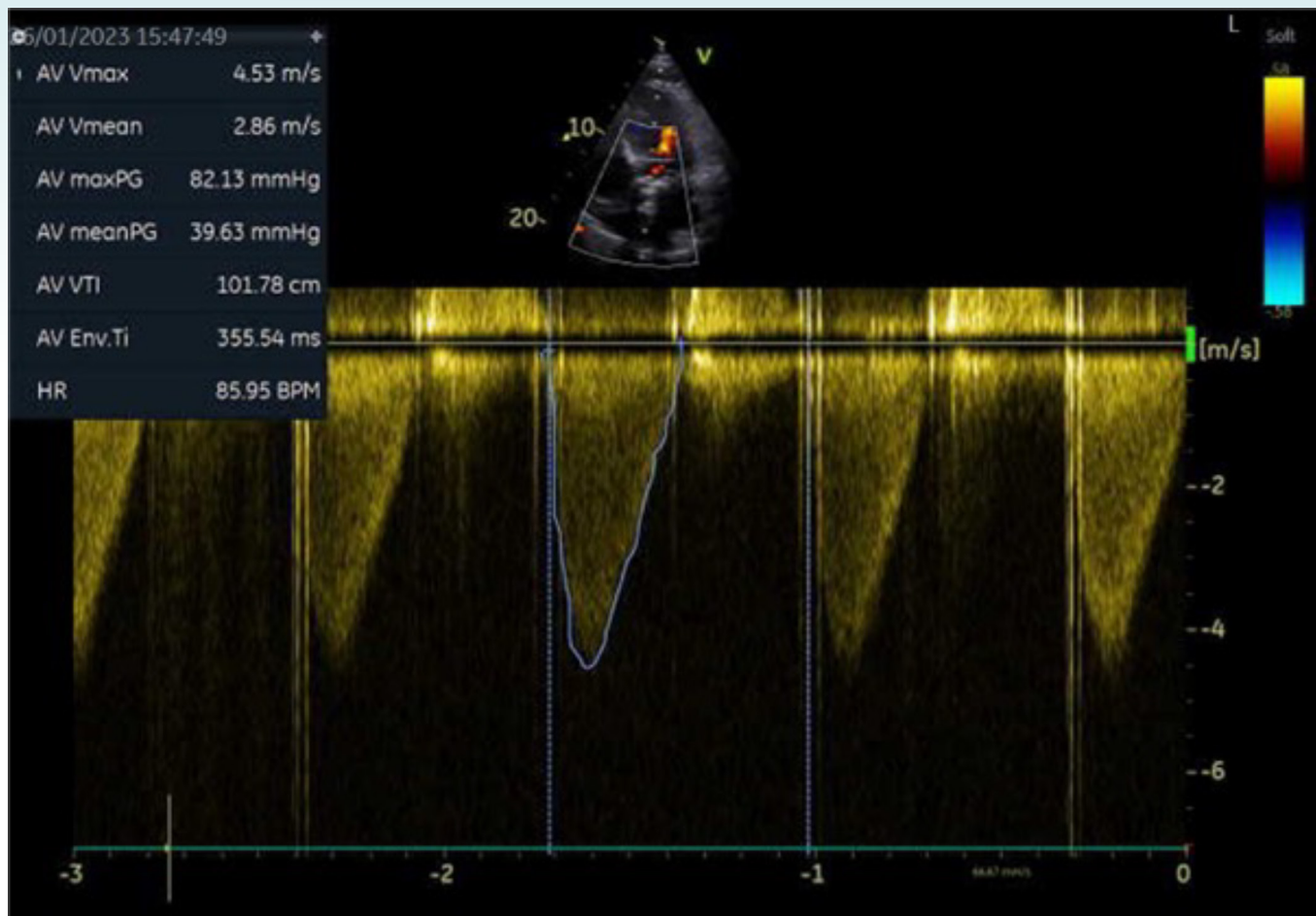
A 67-year-old woman with hypertension, dyslipidemia and paroxysmal atrial flutter was submitted to a mechanical single-tilting disc aortic valve replacement due to severe stenosis in 2009. More recently, she had an history of labile INR, despite therapeutic compliance with warfarin, and an embolic acute myocardial infarction.

In January 2023, she was admitted due to acute onset dyspnea. Physical examination was positive for respiratory distress, bilateral diffuse rales, muted mechanical heart sounds, hypotension and signs of peripheral malperfusion. Initial electrocardiogram revealed sinus rhythm with generalized ST-segment depression. Initial workup showed acute respiratory failure with hyperlactacidemia and an INR of 1.97. Transthoracic echocardiogram (TTE) showed an aortic mechanical prosthetic valve with mean gradient of 45mmHg and free intraprosthetic regurgitation (PHT<100ms) with preserved biventricular systolic function. Rapid ventilatory and hemodynamic deterioration led to invasive ventilation and aminergic

support. Transesophageal echocardiogram confirmed an immobile hemi-disc, in an open position, causing free aortic regurgitation, and an image suggestive of intra-prosthetic thrombus. It was assumed a cardiogenic shock in the context of free aortic valve regurgitation due to mechanical PVT in a patient with infratherapeutic INR. Considering the distance to a surgery centre and the patient's instability, fibrinolysis was started with favorable response in the first 12 hours, allowing suspension of vasopressors and extubation. INR was hardly kept on target range values despite association with acetylsalicylic acid, and a few days later, serial TTEs showed an aortic prosthetic valve with almost normal leaflet motion, mean gradient of 20mmHg, mild prosthetic regurgitation and no images suggestive of thrombus. She was discharged after 12 days and is currently in outpatient follow-up, with no recurrence of events.

DISCUSSION

This case report demonstrates the importance of anti-coagulation efficacy in order to prevent complications, and the need for a high index of suspicion and rapid diagnosis. Surgery is the first treatment of choice but considering the clinical status with cardiogenic shock and the distance from a surgical centre, fibrinolysis played a leading role as a rescue therapy. Despite the favorable evolution, prosthesis replacement might be considered, given the difficulty in achieving stable INR values.



CC 19

“Two is company, three is a crowd” - a case report of cardiac amyloidosis, hypertrophic and ischemic cardiomyopathy

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INTRODUCTION

Differential diagnosis in patients with hypertrophic cardiomyopathy phenotype is crucial. Cardiac amyloidosis has been increasingly diagnosed among older patients. There are no drugs available that result in regression of the disease.

CASE REPORT

A 76-year-old man, good functional status, was referred to our Cardiology appointment. He had an important past medical history with hypertrophic cardiomyopathy genetically confirmed (CSRP3 gene) and previous ischemic cardiomyopathy with implantation of a stent in the median left anterior descending artery, with preserved ejection fraction. He had an implantable cardioverter-defibrillator for primary prevention. Furthermore, atrial fibrillation, arterial hypertension, type 2 diabetes and obstructive sleep apnea were present.

He had complaints of dyspnea for less than ordinary activity (New York Heart Association Functional Classification [NYHA] III) which improved after increasing oral furosemide. N-terminal brain natriuretic peptide levels also improved after this adjustment (19600 to 5570 pg/mL) and the patient remained stable in NYHA II. However, a routine transthoracic echocardiogram revealed severe ventricular hypertrophy with granular sparkling of myocardium, reduced global longitudinal strain despite preserved left ventricular ejection fraction, low myocardial velocities, reduced longitudinal function of right ventricle, atrial enlargement, calcified sub-valvular aortic and

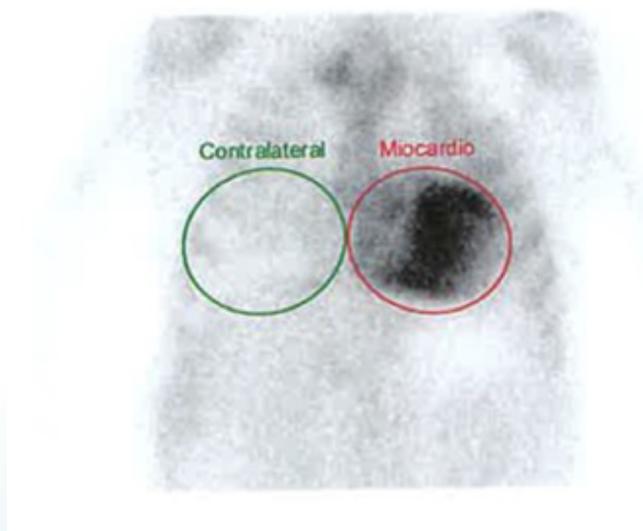
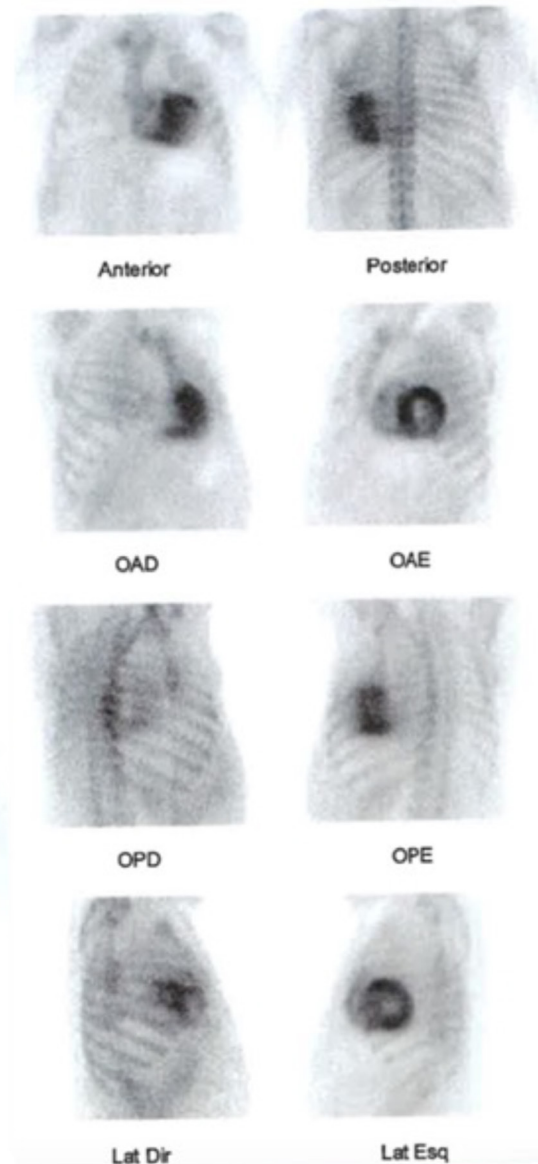
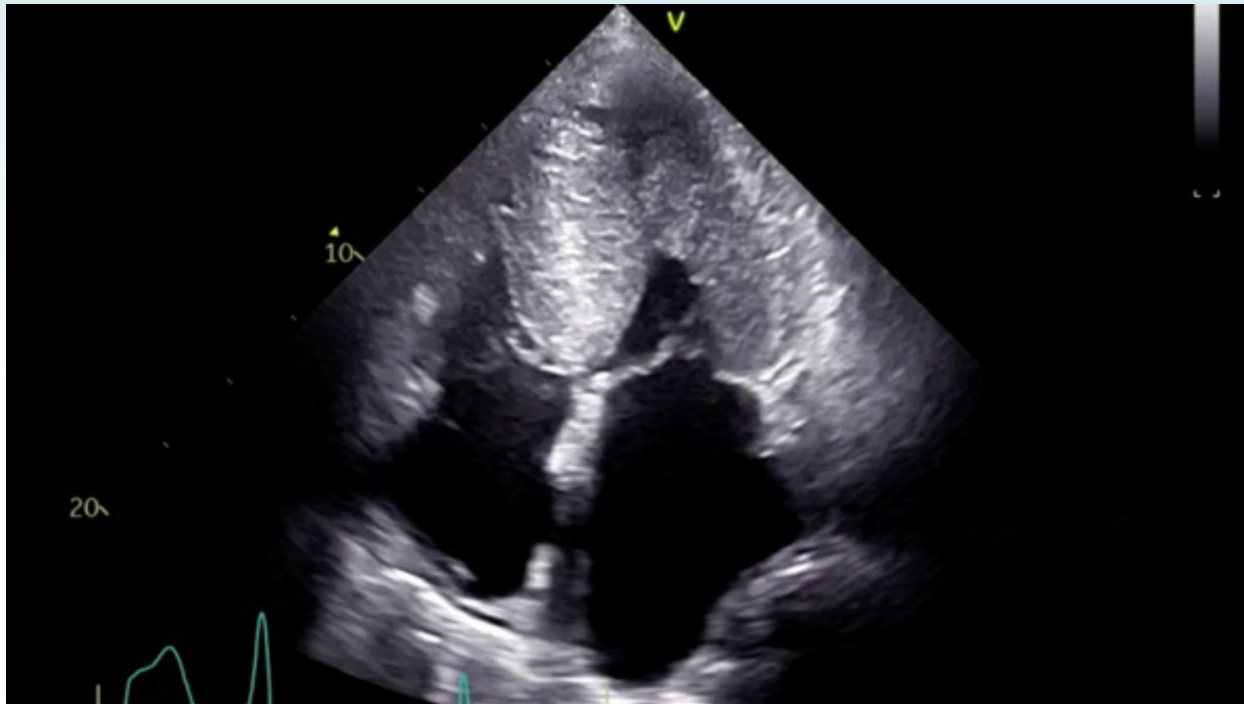
mitral apparatus and mild pericardium effusion - which were suspicious for cardiac amyloidosis. Immunofixation and free light chain assay were negative and bone tracer cardiac scintigraphy (99mTc 3,3-diphosphono-1,2-propanodicarboxylic acid) positive (grade 3). Genetic testing was negative, confirming wild-type transthyretin (ATTR) amyloidosis, not eligible for tafamidis due to extension of disease and patient's comorbidities.

Several months later and despite optimized medical therapy the patient needed inpatient care for two worsening heart failure events (one month apart) due to progression of his disease. The patient clinically improved after intravenous diuretics and was discharged after 6 and 7 days, respectively. In the second event, thoracentesis was performed due to persistent right pleural effusion, with no other intercurrents registered.

Since the patient was discharged he remains in stable NYHA II. Nevertheless, he needs regular outpatient management with intravenous diuretic administration.

DISCUSSION

This case highlights the importance of differential diagnosis in patients with hypertrophic cardiomyopathy phenotype to precociously identify and treat cardiac amyloidosis. Our patient had both confirmed genetic mutation for hypertrophic cardiomyopathy and wild-type ATTR amyloidosis, a rare association which makes this case a diagnosis and treatment challenge.



CC 21

Keep Left, Pace Right

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INTRODUCTION

The venous access to pacemaker lead implantation implies the evaluation of the venous pathway from the upper limb to the right heart chambers. Persistent left superior vena cava (PLSVC) is the most common disorder of the venous system but still a rare congenital disorder that is mostly asymptomatic but may occur in association with other congenital heart abnormalities/syndromes.

It is mostly discovered while performing interventions through the left subclavian vein or through a dedicated thoracic imaging.

CLINICAL CASE

A 78-year-old man with excellent functional status with hypertension and paroxysmal atrial fibrillation and a persistent left bundle branch block presents to the emergency department with dizziness and extreme fatigue noted over the course of one week.

The physical exam shows bradycardia and the ECG documents a complete heart block with an escape rhythm of about 35 bpm with a right bundle branch morphology. He was admitted to the Cardiology department for vigilance and definitive device implantation. Due to the patient's functional status he was a candidate for eventual CRT-P implantation if he presented left ventricular dysfunction.

The pre-implant echocardiogram showed a normally sized heart chambers with a dilated coronary sinus (35x18 mm), a moderate concentric left ventricular hypertrophy and a normal left ventricular function (LVEF 69%). Because of this, the chosen device was a dual chamber pacemaker.

In the pacing room the cephalic vein was isolated and due to an anomalous lead progression, i.e. a descending orientation along the left side of the chest and not crossing the midline, we decided to perform a venography that showed a persistence of the left superior vena cava with drainage in the coronary sinus.

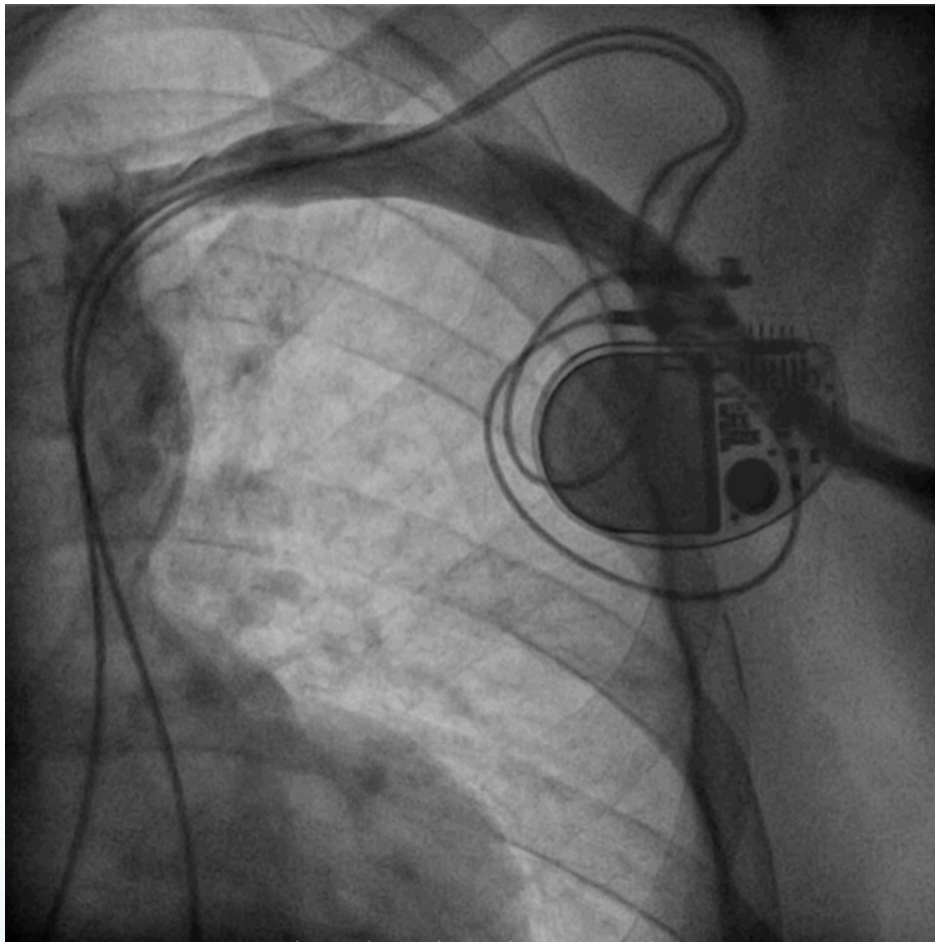
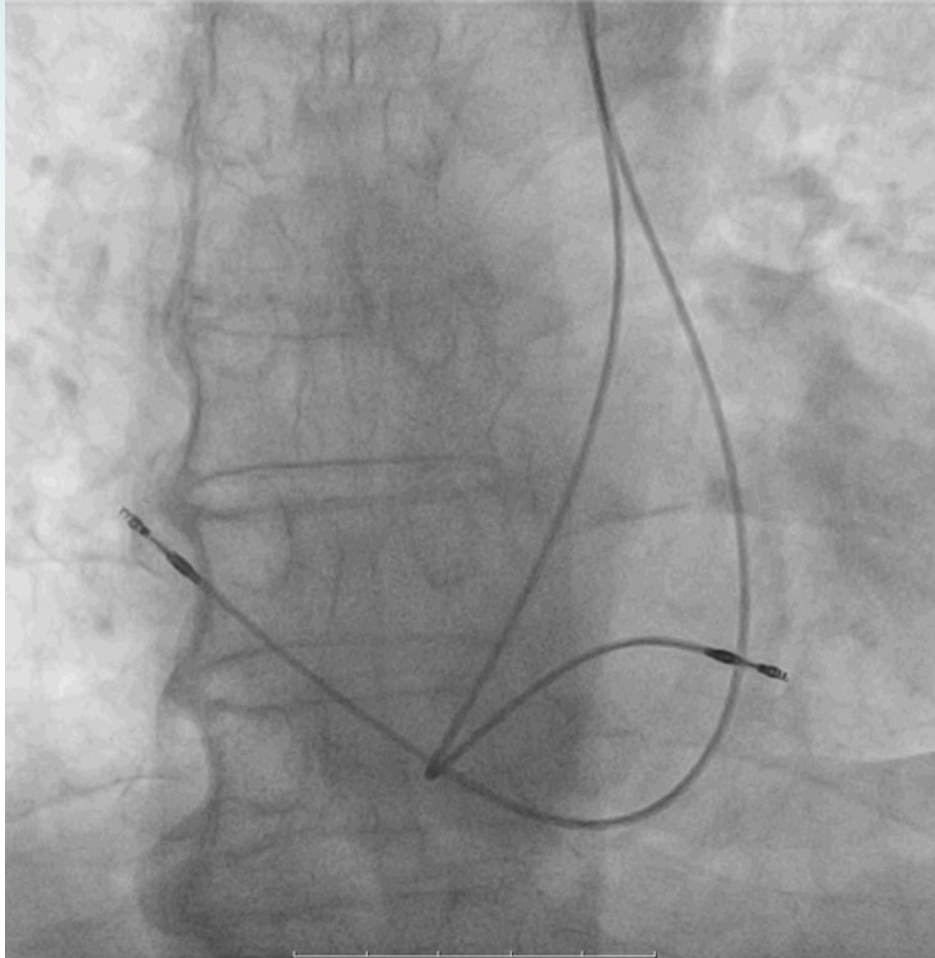
There was difficulty in passing the lead to the right ventricle but, despite this, there were good sensing and pacing parameters. The ventricular active fixation lead was placed in the middle interventricular septum.

DISCUSSION

PLSVC mostly an asymptomatic condition though there has been reports of patient with atrial fibrillation in whom there were identifiable sources of ectopy in the left persistent vena cava.

PLSVC can be suspected or even diagnosed with transthoracic echocardiography but, in most cases, it is diagnosed with transesophageal echocardiography, venography or computed tomography.

In pacemaker implantation, this anatomical variant causes a problem in the placement of the ventricular lead due to the acute angle to enter the right ventricle. Different shapes can be imposed to the stylet to help in this motion and reduce the probability of a complication, and if the left side cannot be used safely, a right sided approach should be attempted.



CC 22

No Access

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INTRODUCTION

The implantation of a pacemaker requires seldom an extensive evaluation of the vascular accesses to plan the procedure. In a patient with pacemaker indication, the presence of arteriovenous fistula is usually seen as indication to implant the device contralateral in order to avoid central venous stenosis that may result in limb and breast pain and eventually arteriovenous access failure. A leadless pacemaker does not present this problem.

CLINICAL CASE

An autonomous 83-year-old woman, in regular hemodialysis secondary to a granulomatosis with polyangiitis presents to the emergency department at night with an episode of sudden loss of consciousness with resulting head trauma.

She showed no signs of hypoperfusion, had normal blood pressure, and a heart rate of about 30 bpm. The ECG showed complete AV heart block with a complete right bundle branch block. Blood work was unremarkable. A fast echocardiogram showed normal biventricular function and an aortic sclerosis. The cranial TC showed raised the suspicion of a thin layer of acute subdural hematoma.

It was decided to implant a temporary transvenous pacemaker. The patient had a right femoral arteriovenous prosthesis and thus the lead was implanted through the left femoral vein after an echo-guided puncture. After this, the patient became pace dependent until dialysis, after which she transitorily recovered sinus rhythm.

Given the need to reevaluate the cranial CT findings, we also requested an angio-CT scan to evaluate the permea-

bility of the venous accesses for a conventional pacemaker. It showed no signs of acute complication from the head trauma and also a marked reduction of the caliber of the brachiocephalic trunk as well as filiform right subclavian and right internal jugular. She had additionally non-working arteriovenous fistulas in both arms.

She was elderly and on a regular dialysis program, so we decided to implant a leadless system with atrial sensing capability. But first, a new transvenous temporary pacemaker had to be placed through the permeable left internal jugular vein to then remove the first temporary femoral lead in order to use the left femoral access to deliver the leadless pacemaker.

Though technically more challenging, the left femoral venous access was used with success. The AV leadless pacemaker was placed in the middle septum. After the procedure, the pacemaker showed a very significant AV synchrony.

DISCUSSION

The leadless pacemaker is a self-contained system implanted directly into the right ventricle, delivered via a femoral vein transcatheter approach. The primary advantage of a leadless pacemaker is the elimination of several complications associated with transvenous pacemakers and leads, namely infections - particularly relevant in dialysis patients. The battery life comparable to that of a transvenous pacemaker, and at end of battery life it can be turned off and a new pacemaker implanted.

CC 25

Thoracic and cardiac masses: different locations, same etiology

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Diffuse large B-cell lymphoma is the most common histologic subtype of non-Hodgkin lymphoma. Involvement of the heart can be seen late in advanced cases, but it is uncommon for diffuse large B-cell lymphoma to present as intracardiac mass.

A 67-year-old female, with past medical history of hypertension, dyslipidemia and obesity, came to the emergency department due to exertional dyspnea, orthopnea and pain in the posterior left thoracic region. At admission, BP was 156/95mmHg, temperature 36.3°C and oxygen saturation was 97%. Physical examination revealed decreased breath sounds at the left lung base, bibasilar crackles and bilateral ankle edema. Initial ECG showed sinus rhythm, 98/min and S1Q3T3 pattern and lab testing revealed Hb 11.30g/dL, 538.000 platelets and elevated pro-BNP and d-dimers, with no other abnormalities.

Chest CT showed a 12.5cm intramuscular dorsal mass in the left posterior region of the chest wall, with invasion of the adjacent 6th and 9th costal arches and an endoluminal image in the right atrium, with contrast uptake. Transthoracic echocardiography with suboptimal acoustic window revealed moderate thickening of the interventricular septum and right atrial wall, globally preserved biventricular systolic function and low volume circumferential pericardial effusion, without significant hemodynamic compromise. Intracardiac mass described in CT was not clearly visualized.

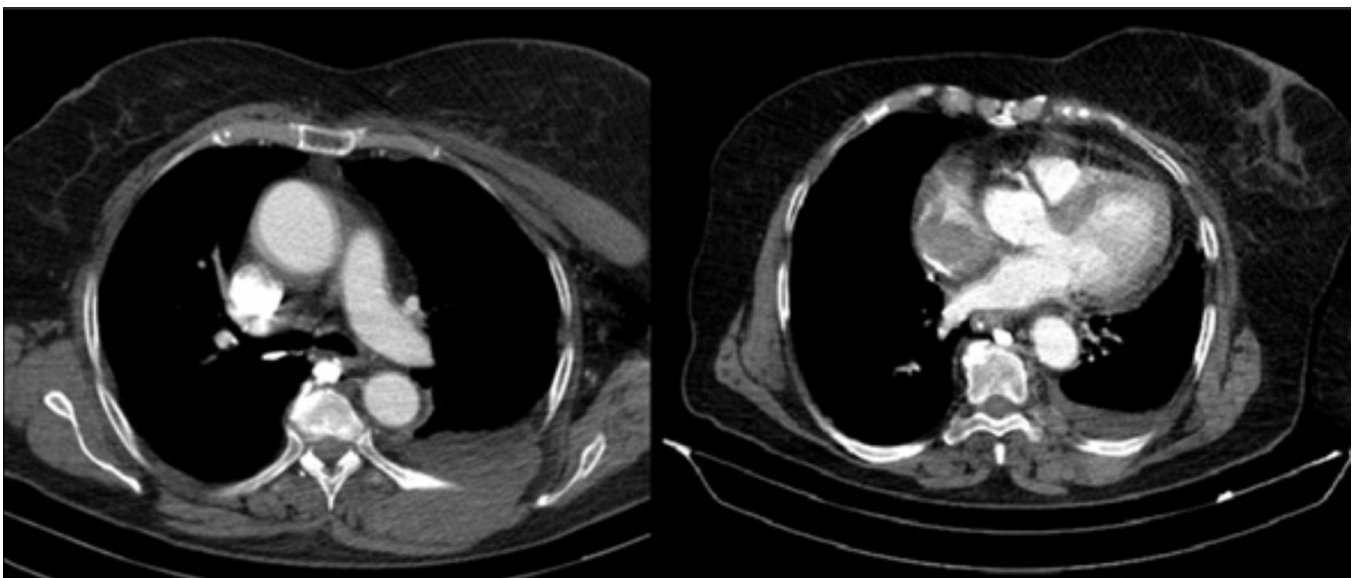
A cardiac magnetic resonance was performed to better assess the cardiac mass. It confirmed the presence of a lesion in the right atrium, extending superiorly between the plane of the superior vena cava and the ascending aorta, and ending above the level of the pulmonary artery and another lesion close to the right atrioventricular sulcus. These lesions were hyperintense in T2 and isointense in T1 (without fat saturation), with perfusion and heterogenous early and late gadolinium enhancement, suggestive of malignant neoformation.

A chest wall bone biopsy was taken and histopathology revealed high degree non-Hodgkin lymphoproliferative process with characteristics of diffuse large B-cell lymphoma. The patient started chemotherapy with progressive clinical improvement.

Revaluation CMR 7 months later revealed almost complete resolution of the cardiac mass and thoraco-abdominopelvic CT showed significative reduction of the lesion on the left posterior intercostal space. PET scan showed no evidence of hypermetabolic lymphomatous disease.

She is clinically stable and maintains follow-up in hematology and cardio-oncology consultation.

This case illustrates a favorable clinical course of a diffuse large B-cell lymphoma with cardiac involvement and highlights the importance of multimodality imaging characterization in this rare condition. A high index of suspicion and proper investigations are recommended to allow for early diagnosis and intervention.



CC 25

Stent-a-cular: How IVUS-guided covered stent angioplasty defeated a massive LAD coronary artery aneurysm

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INTRODUCTION

Coronary artery aneurysm (CAA) is a rare finding during angiography and its underlying causes are not fully understood. In adults, atherosclerosis is the primary contributing factor. There is currently no standard protocol for treating CAA. This case report showcases the effectiveness of using PCI to manage a giant CAA.

CASE STUDY

We present the case of a 71-year-old male who was admitted to the Emergency Department due to new-onset of chest pain at rest. He had a medical history of two myocardial infarctions. During coronary angiography, we identified a large saccular proximal left anterior descending coronary artery aneurysm. The patient underwent successful covered stent angioplasty guided by intravascular ultrasound, resulting in a positive outcome.

DISCUSSION

The management of CAA is a complex and individualized process that considers various factors such as aneurysm characteristics, technical considerations, and the patient's clinical status. Further research is required to determine the efficacy of using PCI as a treatment option for CAA.

CC 27

Patent foramen ovale (PFO) and cryptogenic stroke

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INTRODUCTION

PFO is the most common congenital heart abnormality of fetal origin and is present in ~25% of the worldwide adult population. In most cases, PFO is entirely benign and requires no treatment. However, it may cause serious complications under certain circumstances.

CLINICAL CASE DESCRIPTION

A 47-year-old female, caucasian, with no previous medical history, no smoking history and only medicated with a combined oral contraceptive (COC), presented to the Emergency Room after a sudden onset of prostration, left side hemiparesis and right conjugate eye deviation. Initial evaluation revealed head CT and angio-CT changes consistent with middle cerebral artery (MCA) - M1 ischemic stroke. With a NIH Stroke Scale (NIHSS) score of 19, the patient was submitted to fibrinolysis with intravenous recombinant tissue plasminogen activator (rtPA) and later mechanical thrombectomy. Transcranial Doppler sonography and transesophageal echocardiography (TEE) were conducted to assess possible cardiac etiology leading to diagnosis of a PFO associated with moderate right-to-left shunt after Valsalva maneuver. Thrombophilia testing revealed a heterozygous mutation of MTHFR A1298C gene. Antiphospholipid antibodies were within normal ranges. A vitamin B12 deficiency was also present. Cardiology evaluation revealed no further abnormal findings. Months later she was submitted to percutaneous PFO closure with a 25mm Amplatzer PFO Occluder without complications.

DISCUSSION

Elevated homocysteine levels are a risk factor for developing blood clots. If a person is deficient in vitamin B12, B6 or folate, homocysteine cannot be efficiently recycled. The enzyme methylenetetrahydrofolate reductase (MTHFR) is also involved in this process. Inherited mutations of the MTHFR gene are very common and may lead to an enzyme that is not optimally active and, consequently, to elevated homocysteine levels. Finding elevated levels doesn't influence management. The MTHFR mutations by themselves, in the absence of elevated homocysteine levels, are not a risk factor for cardiovascular disease or venous thromboembolism in countries where food is fortified with folic acid. They are not clotting disorders. PFO is the consequence of failed closure of the foramen ovale, a normal structure in the fetus to direct blood flow directly from the right to the left atrium, bypassing the pulmonary circulation. Its association with an increased risk of stroke is attributed to the paradoxical embolism of venous thrombi that shunt through the PFO directly to the left atrium. Although the risk of stroke recurrence is considered low, considerable controversy remains regarding the optimal treatment strategy for patients with both cryptogenic stroke and PFO. Individually, PFO, COC use and MTHFR mutations are low risk factors but together they might form a dangerous combination.

CC 28

Riding the Waves: Tackling Thyrotoxic Crisis and Atrial Fibrillation Challenges

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BACKGROUND

Thyrotoxicosis is a medical condition characterized by the excessive production of thyroid hormones as a result of an overactive thyroid gland. It represents a significant risk factor for the development of atrial fibrillation (AF) due to its pronounced impact on electrical impulse generation and conduction. This pathological influence is associated with a shortened action potential duration and contributes to the occurrence of AF.

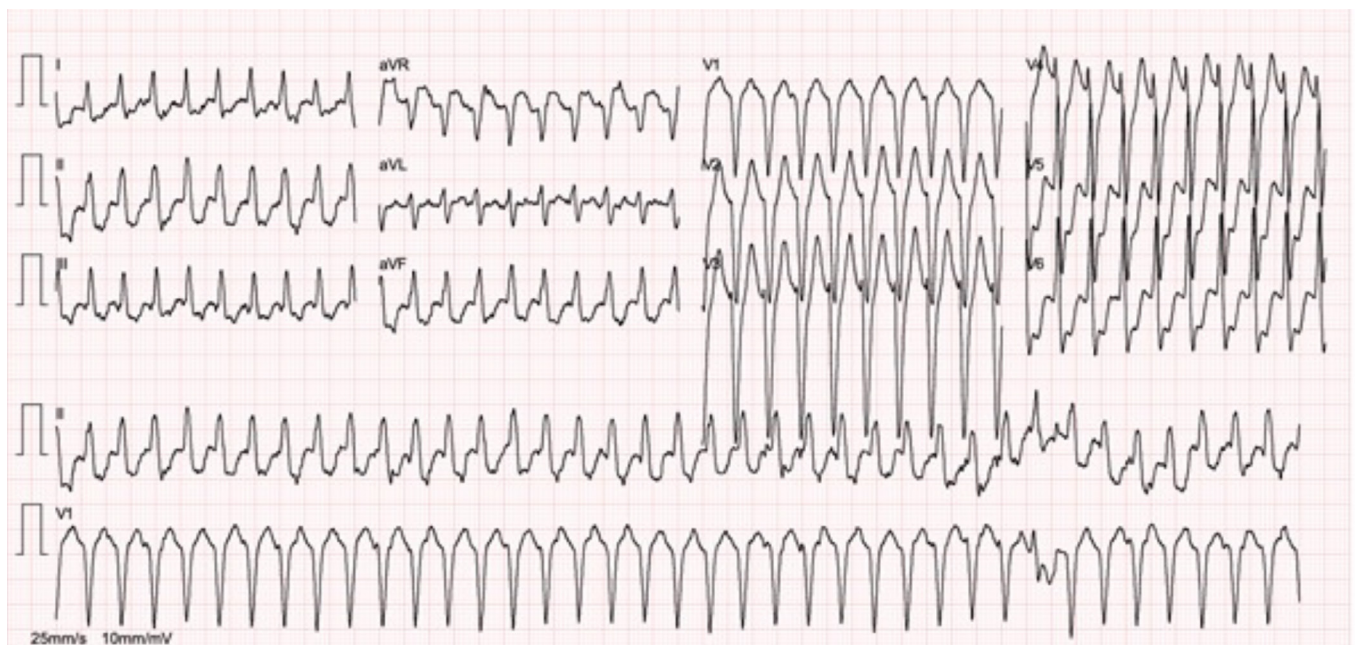
CASE

In this report, we present the case of a 74-year-old male patient who was admitted to the emergency department exhibiting symptoms including lethargy, fever, anorexia, weight loss, fine tremors, and tachycardia. His initial vital signs recorded a blood pressure of 129/75 mmHg, a heart rate of 220 beats per minute, a body temperature of 38.2°C, and a respiratory rate of 25 breaths per minute. An electrocardiogram (ECG) analysis revealed a rapid ventricular response indicative of atrial fibrillation. Upon physical examination, the patient presented with a fever of 38.2°C and exhibited a severe comatose state. Laboratory investigations unveiled an undetectable level of thyroid-stimulating hormone (TSH) (<0.01 [ref: 0.34 - 5.60] µUI/mL) and an elevated level of free T4 (>77.8 [ref: 7.9 - 14.4] pmol/L). White blood cell count and liver function tests yielded normal results. The patient was diagnosed with

thyroid crisis accompanied by atrial fibrillation with a rapid ventricular response. Prompt intervention was initiated, comprising the administration of intravenous propranolol (10mg), hydrocortisone (100mg), and thiamazole (10mg). The patient demonstrated a favorable response, as evidenced by a reduction in heart rate to 110 beats per minute. However, despite these measures, the patient's condition deteriorated several hours later, culminating in a cardiopulmonary arrest characterized by asystole, which proved refractory to advanced life support measures.

CONCLUSION

Thyroid storm is associated with a mortality rate ranging from 10% to 20%, often attributed to the challenges posed by tachyarrhythmias that emerge in the context of hyperthyroidism. Initial management of tachycardia typically involves the use of beta-blockers and antithyroid therapy, with the avoidance of amiodarone due to concerns regarding its potential exacerbation of hyperthyroidism. This case serves as a poignant reminder of the importance of considering thyroid crisis within the differential diagnosis of patients presenting with similar clinical manifestations. Furthermore, it underscores the reality that even with the administration of seemingly appropriate and effective treatments, patients may still experience decompensation.



CC 29

Unveiling the Enigma: Takotsubo Cardiomyopathy and Wellens' Syndrome - A Complex Case Illustrating Diagnostic Dilemmas in ECG Interpretation

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BACKGROUND

Takotsubo cardiomyopathy, also referred to as apical ballooning syndrome and stress cardiomyopathy, is a transient condition characterized by systolic and diastolic dysfunction of the left ventricle, resulting in various abnormalities in cardiac wall motion. This condition is increasingly associated with significant morbidity and mortality. Wellens' syndrome is an electrocardiographic (ECG) pattern observed in asymptomatic patients that indicates critical occlusion of the left anterior descending coronary artery, necessitating immediate cardiac catheterization.

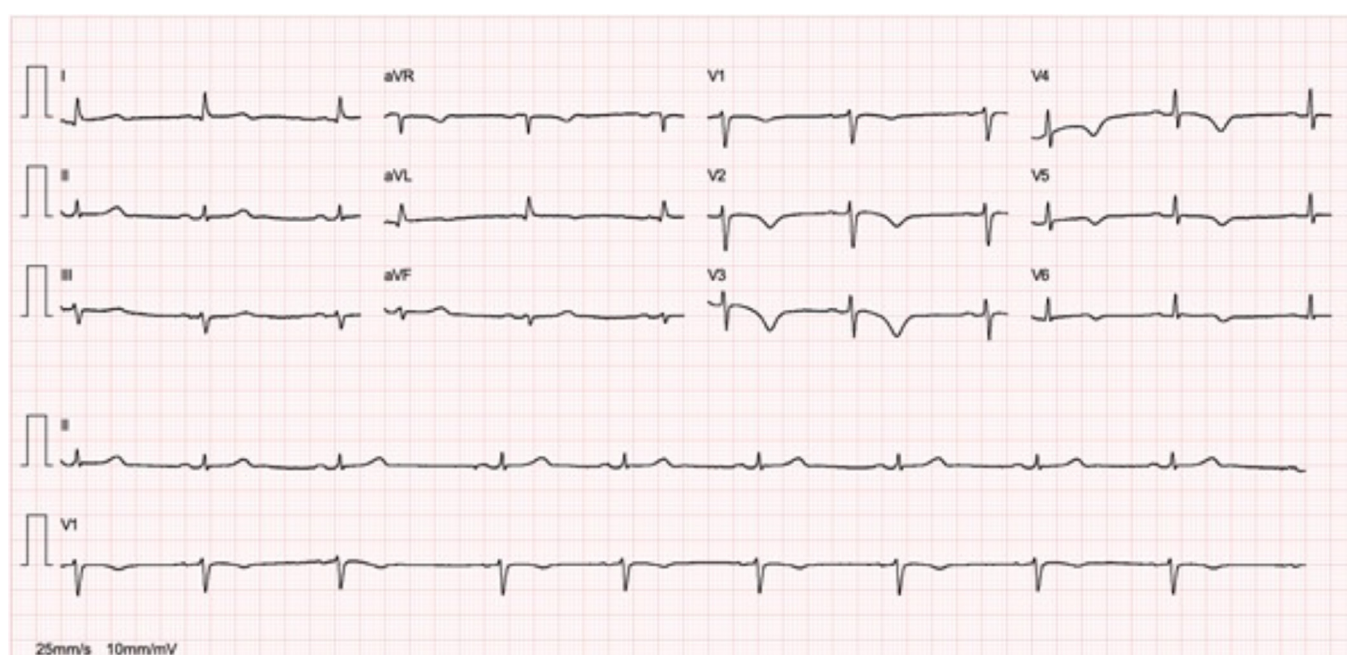
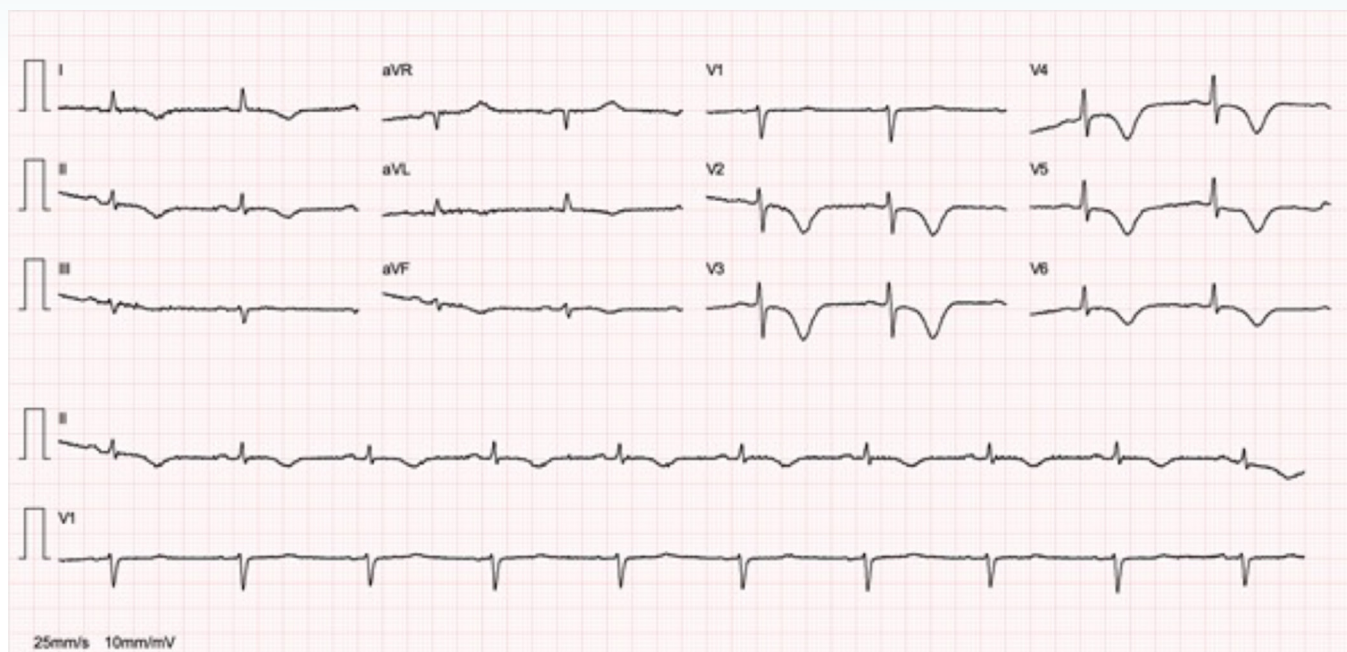
CASE REPORT

A 61-year-old woman with a medical history of paroxysmal atrial fibrillation, mixed hyperlipidemia, and hypothyroidism presented to our emergency department with intermittent retrosternal chest pain and dyspnea on exertion persisting for approximately one week. The chest pain was described as a dull, pressurizing sensation, and the initial troponin level was elevated at 1986 pg/dL (reference range <20 pg/dL). The initial 12-lead electrocardiogram (EKG) exhibited significant T-wave inversions extending across the precordial leads. The patient was administered aspirin and unfractionated heparin, and subsequently transferred to our facility for evalua-

tion of non-ST-segment elevation myocardial infarction. Upon arrival, a subsequent 12-lead EKG was performed, revealing dynamic changes in the precordial leads with more pronounced T-wave inversions, as well as new inversions across leads II, I, and aVL (Figure 2). The patient underwent cardiac catheterization, which demonstrated angiographically normal coronary arteries, moderately impaired left ventricular function with an ejection fraction (EF) of 35%, and wall-motion abnormalities, specifically dyskinesia of the apical anterior wall and apex observed on left ventriculogram. No evidence of coronary disease was found. These findings were consistent with the diagnosis of Takotsubo cardiomyopathy. Transthoracic echocardiography confirmed apical dyskinesia consistent with Takotsubo cardiomyopathy. The patient was subsequently discharged with plans for outpatient follow-up, including repeat EKGs, goal-directed medical management of heart failure with reduced ejection fraction, and repeat transthoracic echocardiography.

DISCUSSION

This case underscores the importance of considering a broader differential diagnosis when encountering ECG findings suggestive of an equivalent of ST-segment elevation myocardial infarction (STEMI).



CC 31

A Right-sided Obstruction

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INTRODUCTION

Cardiac malignant neoplasms are more often the result of metastases or direct invasion of the heart. Leiomyosarcoma are a rare primary cardiac malignancy.

CLINICAL CASE

A previously asymptomatic active smoker 62-year-old man, presented to the emergency department complaining of a weeks-long chest and left shoulder pain, without respiratory variation. He reported no accompanying symptoms. He was normotensive with normal heart sounds. The ECG was of sinus tachycardia with an inverted T wave V1-V5. The chest radiography showed an enlarged cardiac silhouette. The bloodwork showed thrombocytopenia, normal white blood cell count and a significantly elevated C-reactive protein.

The chest TC presented a 20 mm circumferential pericardial effusion and a hypodense image in the heart base without other suspicious images. The angiographic phase showed no perfusion defects and normal size mediastinal lymph nodes. An echocardiogram was performed in the emergency department - it showed a small pericardial effusion, a severe dilation of the right-side chambers and diastolic septal flattening suggesting right-side overload, tricuspid annular dilation resulting in severe tricuspid regurgitation; it confirmed a large multilobulate friable intracardiac mass in the internal face of the right ventricle outflow tract (RVOT) causing flow obstruction (maximum RVOT gradient of 35 mmHg).

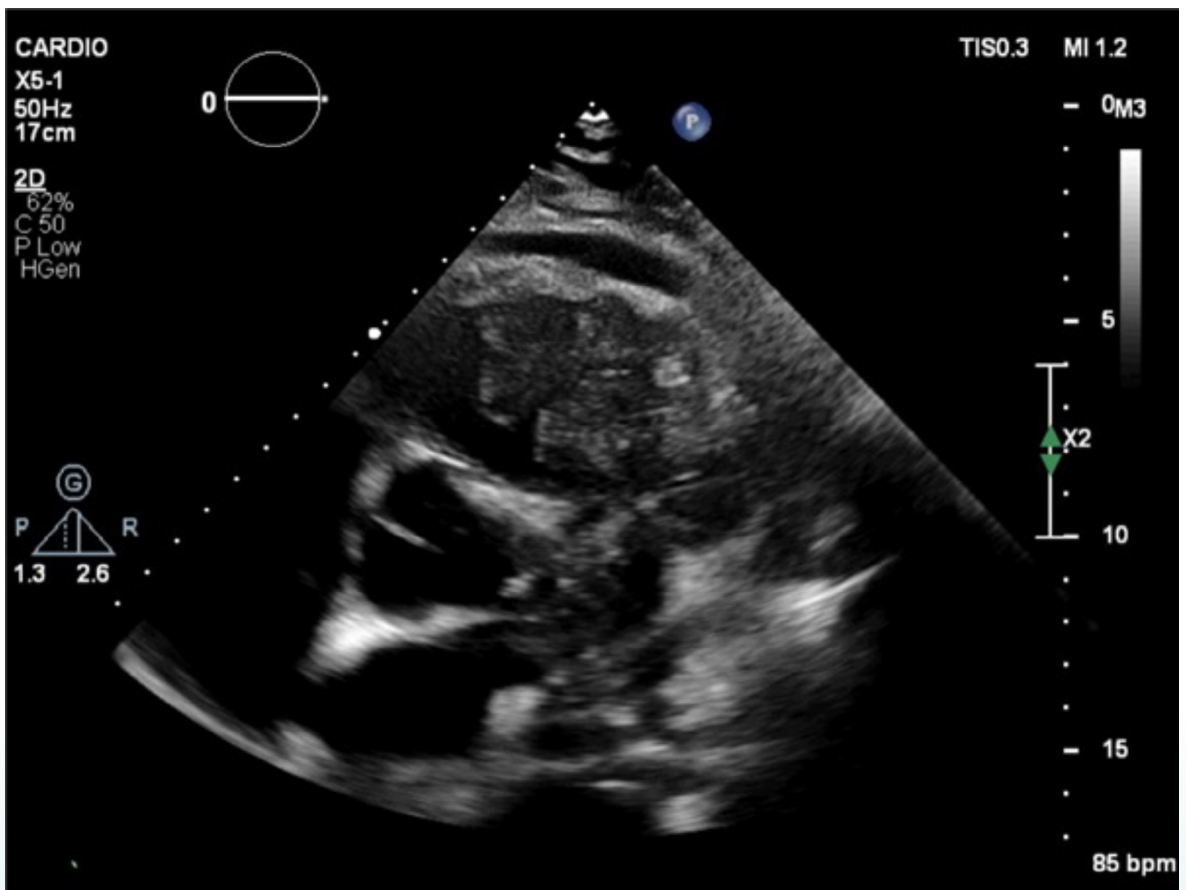
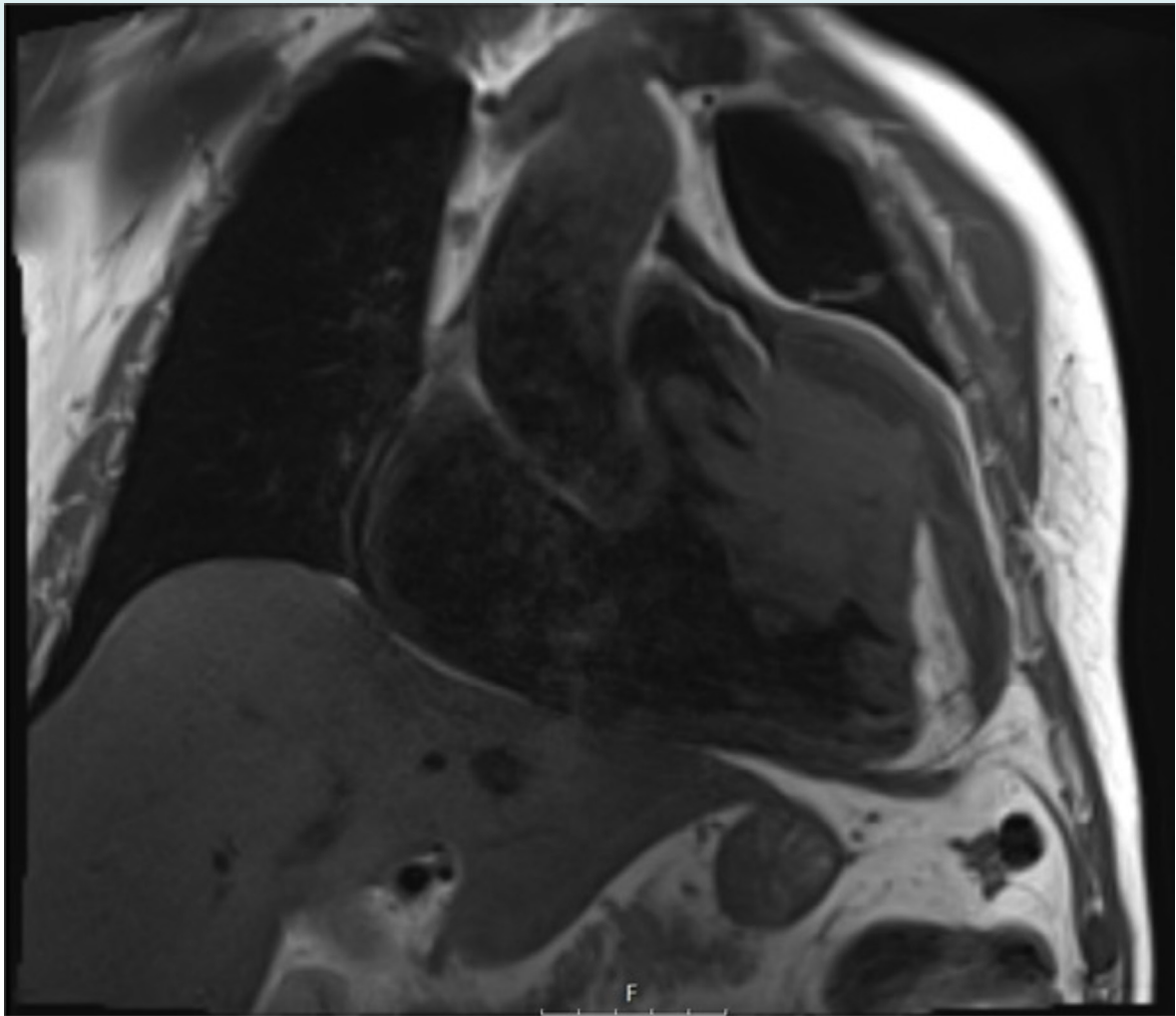
He was admitted to Cardiology. A more dedicated CT was requested: no other masses were evidenced and

the cardiac mass appeared to originate in the pulmonary artery (PA) with local invasion of the heart, raising the suspicion of a PA angiosarcoma. A cardiac MRI was requested for better characterization: it showed a mass of 85 X 65 mm in the RVOT/PA without a cleavage plane and possible invasion of the parietal pericardium; signal heterogeneity suggestive of necrosis and associated thrombus. It was isointense in T1 and hyperintense in T2-weighted image without suppression in fat saturation sequences; it presented diffuse heterogeneous early and late gadolinium enhancement; the pericardial effusion was small with signal heterogeneity suggestive of fibrin or hematic content. To try to establish a diagnosis, the patient was referred for endomyocardial biopsy - the fragments showed a spindle-celled neoplasia with markers of leiomyosarcoma.

Palliative care was decided in a sarcoma's specialized group meeting and the patient was discharged as per his will. Shortly after, the patient presented to a secondary emergency department with an acute episode of dyspnea that progressed to a cardiorespiratory arrest and death.

DISCUSSION

Leiomyosarcomas occurs mostly in the left heart, often with pulmonary veins of mitral valve involvement. The MRI appearance is characteristic but not specific. This is an aggressive tumor with a fast growth, high rate of distant metastases and local recurrence after removal.



CC 32

ASSYMPTOMATIC ruptured sinus of Valsalva aneurysm as a cause of aorto-atrial fistula - A case report

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INTRODUCTION

The sinus of Valsalva aneurysm (SOVA) is a rare condition among the general population and even among all congenital cardiac defects. SOVA is a congenital or acquired discontinuity between the aortic valve annulus and the aortic media caused by weakness or degeneration of the elastic connective tissue. Valsalva aneurysms are usually silent and found incidentally by imaging. On the other hand, ruptured SOVA, a rare event, usually leads to heart failure symptoms and carries a poor prognosis if left untreated.

CLINICAL CASE

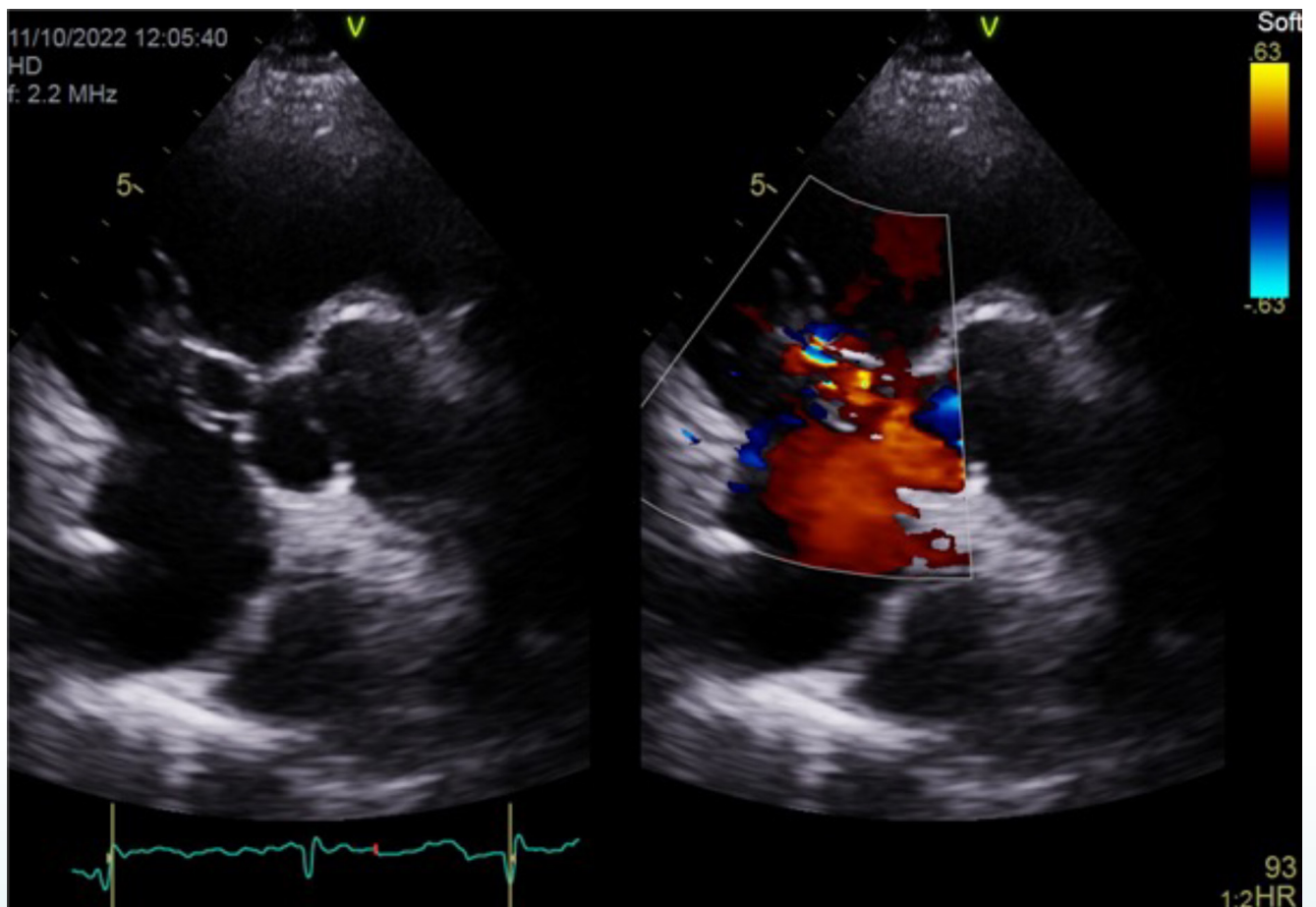
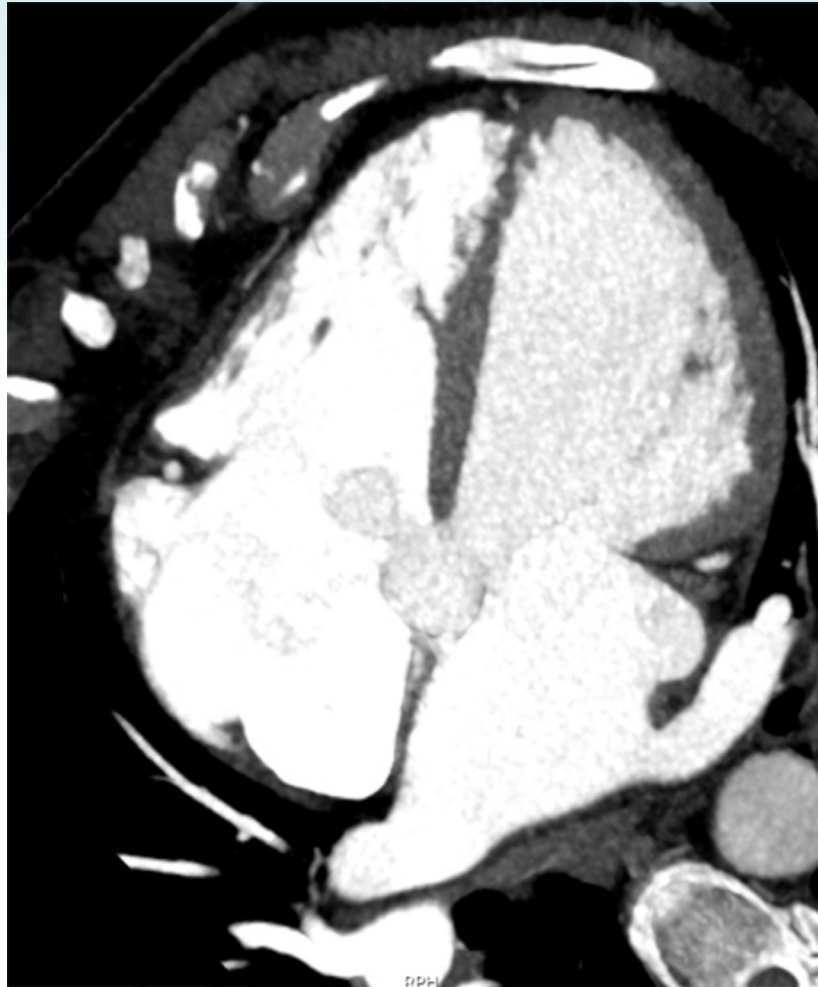
We present a case of a 65-year-old male who performed a transthoracic echocardiogram in the context of the study of a transient ischemic attack. A noncoronary sinus of Valsalva aneurysm/pseudoaneurysm was observed and the color Doppler by transthoracic (fig. 1) and transesophageal echocardiography revealed a significant shunt flow from aorta exiting in direction to the right atrium, confirming the existence of an aorto-atrial fistula. The aneurysm may also be seen in CT scan (fig.2). The patient used to run 10km twice a day. Sometimes, he felt a slight chest discomfort when starting a run, but most of the time he had no symptoms. The left and right

heart catheterization showed normal coronary arteries and elevated right sided and pulmonary capillary wedge pressures. He is now waiting for surgical repair of the fistula and remains asymptomatic.

The patient had no prior aortic surgeries. The majority of infections and connective tissue disorders capable of weakening the elastic tissue were excluded and there was no history of chest trauma. The presence of the aorto-atrial fistula along with the evidence of right heart overload reveal signs of some chronicity, which makes the absence of symptoms of heart failure even more unlikely. Doubts about whether it is an aneurysm or pseudoaneurysm will only be clarified during surgery.

DISCUSSION

A ruptured sinus of Valsalva aneurysm has been described as a rare and frequently devastating event. They most frequently rupture into the right atrium or the right ventricle and less frequently into the left-sided chambers. Ruptured SOVA as a cause of aorto-atrial fistula as the one seen in this case is extremely rare and only a few cases have been reported. Furthermore, congestive heart failure symptoms are present most of the time.



CC 33

Don't take Jesus out of my heart - A case report

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INTRODUCTION

Cardiac myxoma (CM) is a rare tumor that arises from pluripotent mesenchymal stem cells. It represents the most common primary cardiac tumor in adults, affecting mainly patients aged over 65. Nowadays, the easy access to echocardiography allows the early diagnosis of this clinical entity, which, together with the aging of the population, has shown a higher incidence of CM than previously described in the literature. Although myxoma usually presents with a benign histology, its anatomical position, along with its size, mobility and associated thromboembolic events, may be life threatening.

CLINICAL CASE

A 78-year-old woman presented to the emergency room with exertional dyspnea and pleuritic chest pain. Her prior medical history anxiety disorder and breast cancer treated with breast-conserving surgery, radio, and chemotherapy. The patient had already been presenting these symptoms for some months, but they were first related to her anxiety disorder.

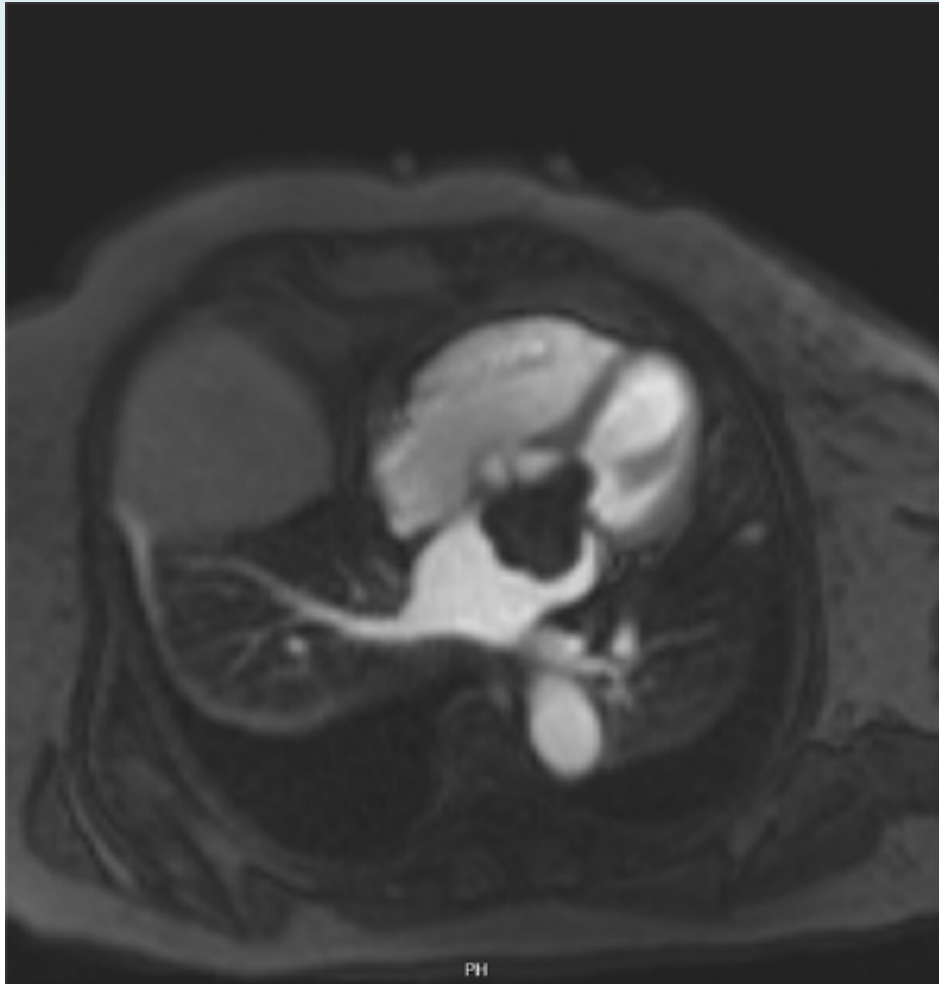
Initial workout revealed high C-reactive protein levels with no further alterations and echocardiography revealed a mass attached to the left side of the interatrial septum with diastolic prolapse into the left ventricle obstructing mitral-valve inflow. Atrial myxoma was suspected. Cardiac resonance imaging confirmed the findings (fig. 1 and 2). The patient's only request was to keep Jesus stay in her heart. Surgical excision was suc-

cessfully performed one week after the diagnosis and histological analysis confirmed the clinical suspicion of cardiac myxoma. The patient was discharged and maintained regular follow-up with echocardiogram, remaining asymptomatic.

DISCUSSION

Clinical presentation of cardiac myxoma is usually unspecific and easily mimic other diseases. When a patient presents to the emergency room with symptoms such as dyspnea, he is not usually evaluated by a cardiologist first. Therefore, the level of suspicion required to reach the diagnosis will have to be higher.

In the presence of heart failure or constitutional symptoms, syncope or embolic phenomena, early performance of transthoracic echocardiography allows to distinguish between cardiac and non-cardiac causes and helps to guide proper diagnosis and treatment. CM might also be considered as a differential diagnosis in the suspicion of valvular heart disease or dysrhythmias. In this case, the elevated C-reactive protein, in the absence of signs of active infection, may be a marker of inflammation, since inflammatory cytokines play an important role in the pathophysiology of CM. Delaying the recognition of CM as a possibility and consequently delaying the performance of an echocardiogram may compromise the outcome, since the atrial myxoma has an unpredictable behavior.



CC 34

Can epilepsy end up in CDI implantation? - A case report

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INTRODUCTION

When approaching an aborted cardiac arrest in young age, cardiac causes must come to mind along with non-cardiac causes. A systematic approach to clinical testing including ECG, echocardiogram, or cardiac magnetic resonance (CMR), exercise test, 24-H Holter monitoring, and drug provocation must be performed. Catecholaminergic polymorphic ventricular tachycardia (CPVT) is a rare hereditary arrhythmogenic disease, whose first presentation may be a sudden cardiac arrest (SCA).

CLINICAL CASE

A 25-year-old boy experienced a cardiac arrest while assisting a football game. Ventricular fibrillation (VF) rhythm was recorded and aborted after electrical defibrillation. His clinical background included epilepsy. There was no family history of cardiac disease or sudden death. Cardiac arrest in the context of epileptic seizure was the first hypothesis. The first 12-lead ECG was normal (fig.1). Echocardiogram and CMR revealed a normal structure and function of the heart and CT coronary angiogram showed normal origin of coronary arteries and absence of coronary heart disease.

An in-depth anamnesis highlighted that diagnosis of epilepsy was made after several absence seizure-like episodes under stress conditions. Previous electroencephalograms never showed abnormal brain electrical activity and cerebral magnetic resonance was free of pathological findings. Furthermore, family history revealed similar episodes in two patients' cousins.

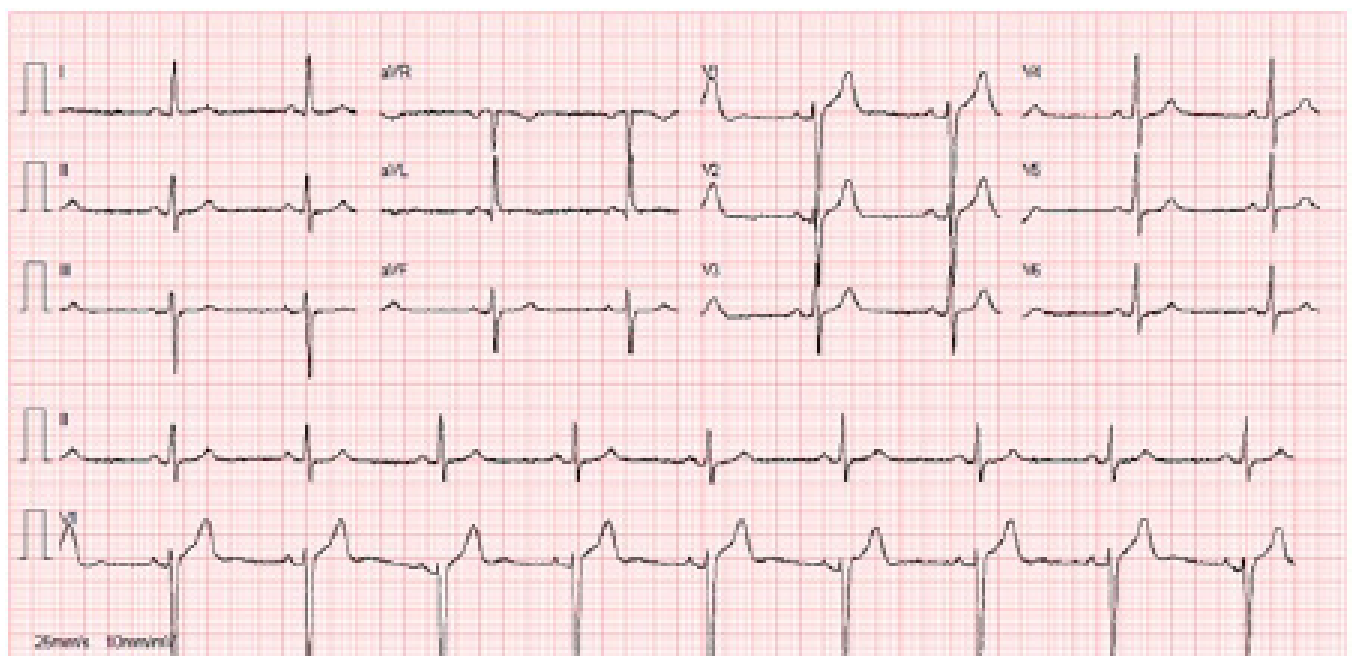
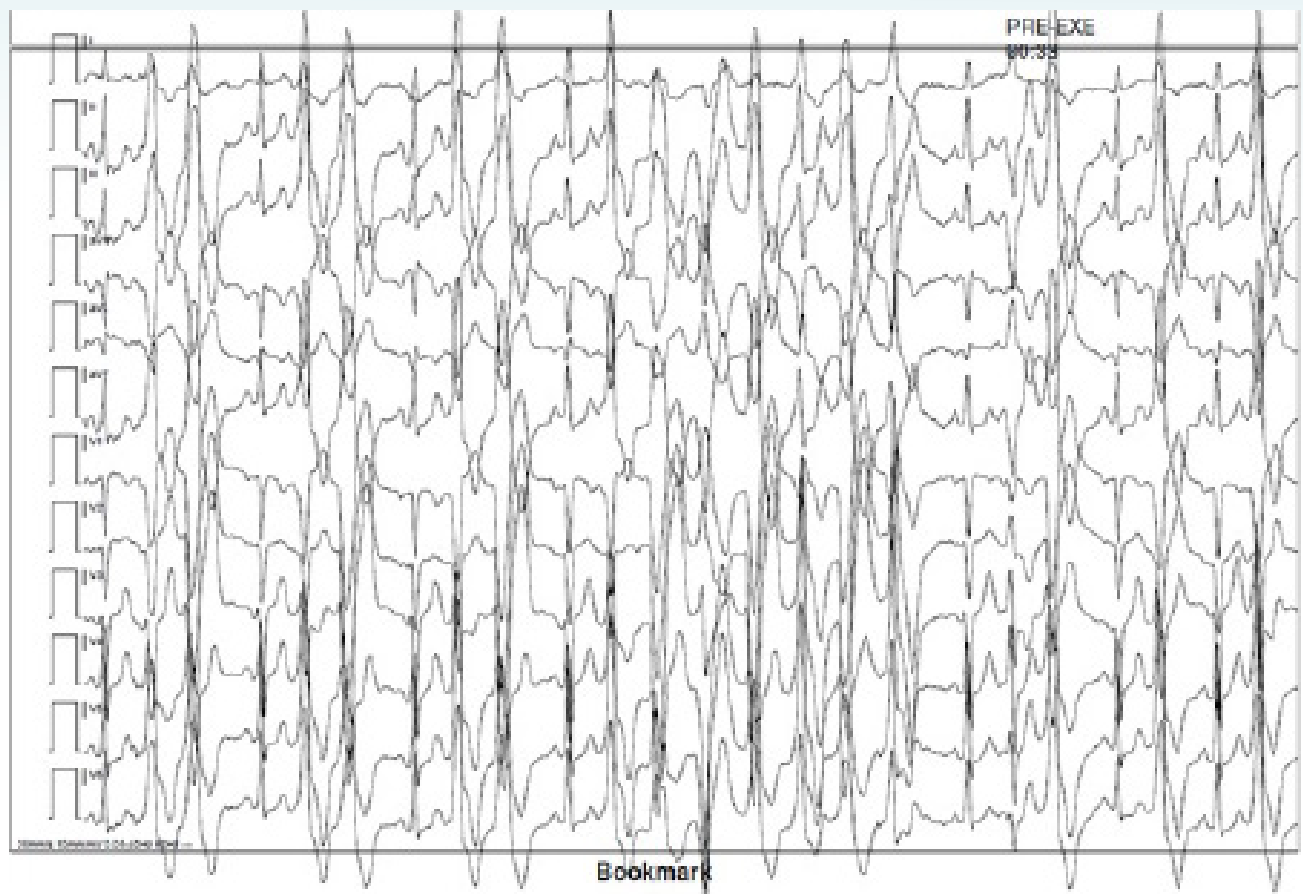
These findings raised the suspicion of an arrhythmogenic disease.

24-H Holter monitoring did not show any arrhythmic event. Provocative testing with ajmaline for Brugada syndrome was negative. An exercise ECG was then performed. Ventricular premature beats early occurred and quickly evolved into a bidirectional tachycardia (fig.2). He was then started on beta-blocker therapy with nadolol and implantation of transvenous implantable cardioverter-defibrillator (ICD) was performed as a secondary prevention intervention. Genetic analysis was performed but the results are not available yet.

DISCUSSION

SCA may be the first presentation of CPVT, a rare and challenging disease. A wrong diagnosis such as epilepsy can delay the final diagnosis and its treatment and allow for serious implications for the patient and his family, with a high mortality rate if left underdiagnosed.

Primary arrhythmic events must be present in the differential diagnosis of absence seizure-like episodes or other non-specific symptoms, particularly when there is no clear evidence of other causes. In the presence of a sudden cardiac arrest in young age, primary arrhythmic event must always be excluded. With this case, we intend to highlight that a more probable diagnosis must not overcome the need for a high suspicion reasoning when the patient fails to meet the expected diagnosis criteria.



CC 35

You can fool the doctor's eyes, but not the CT scan - A case report

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INTRODUCTION

Aortic dissection is an uncommon scenario with a very high mortality rate. Uncontrolled hypertension and atherosclerosis are the main risk factors. It may present as a catastrophic clinical scenario, with chest pain and hemodynamic instability. Here we report a case of an atypical presentation of mildly symptomatic type A aortic dissection.

CLINICAL CASE

A 87-year-old male presented to the emergency department with mild abdominal pain and vomiting for 3 days. His medical background includes hypertension and cerebrovascular disease. On presentation, he was hemodynamically stable and physical examination was remarkable for arrhythmic heart sounds; there were no signs of peripheral hypoperfusion. Blood gas analysis showed increased lactates levels and the diagnosis of *de novo* atrial fibrillation was made by ECG. Laboratory workup revealed elevated levels of myoglobin (4036 ng/mL) and hepatic cytolysis (AST 141 U/L) with no further alterations.

Given the suspicion of mesenteric ischemia, an abdominal CT was performed and revealed a severe type A aortic dissection extending from the aortic root all the way down to the left iliac artery, with aneurysmal dilatation of ascending aorta (7.3cm) - fig. 1 and 2. There were no signs of intestinal ischemia. Then, a transthoracic echocardiogram was performed, confirming the diagnosis and mild aortic regurgitation with no significant pericardial effusion.

Multidisciplinary team, including cardiothoracic surgeon, decide that, considering the age and the comorbidities of the patient and the extension of the dissection, medical treatment was the most appropriate approach. The patient developed heart failure symptoms within a few hours and died the next day.

DISCUSSION

More than 90% of the patients presenting with acute aortic dissection refer severe and sharp chest pain. When patients present asymptotically or with nonspecific mild symptoms as the one here presented, the correct diagnosis is easily missed or delayed. In this case, abdominal pain was probably a symptom of hypoperfusion, as well as *de novo* atrial fibrillation (AF). AF may occur as a consequence of hypoperfusion of sinoatrial nodal artery. It may confound the initial picture, although in this case it contributed for the execution of a contrast-enhanced CT that revealed the final diagnosis.

Mortality rate of type A aortic dissection varies according to age of the patient, gender, and stage at presentation. It is usually an indication for surgery and when medically managed, mortality is significantly higher. For this reason, the choose of medical treatment must be well balanced, considering all the risks and the benefits for the patient. However, the surgical intervention may also be withheld when the impact on survival is not significant since is associated with a high mortality rate *per se*.



CC 36

So you always have been there **- A case report**

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INTRODUCTION

Epidemiology of infective endocarditis (IE) has suffered some alterations given the aging of the population and the increasing use of prosthetic valves and implantable cardiac devices. It remains a rare condition with significant morbimortality.

CLINICAL CASE

A 75-year-old male was admitted by ST-segment elevation myocardial infarction. Transthoracic echocardiogram revealed severe depression of left ventricular systolic function and mild depression of right ventricular function. Emergent cardiac catheterization showed thrombotic occlusion of right coronary artery and primary angioplasty was successfully performed. The patient developed cardiogenic shock, requiring aminergic support, with clinical recovery in the subsequent days.

His past medical history included aortic valve replacement with biologic prosthesis 3 years ago and an implantable pacemaker. He had atrial fibrillation and suffered an embolic stroke 5 months ago.

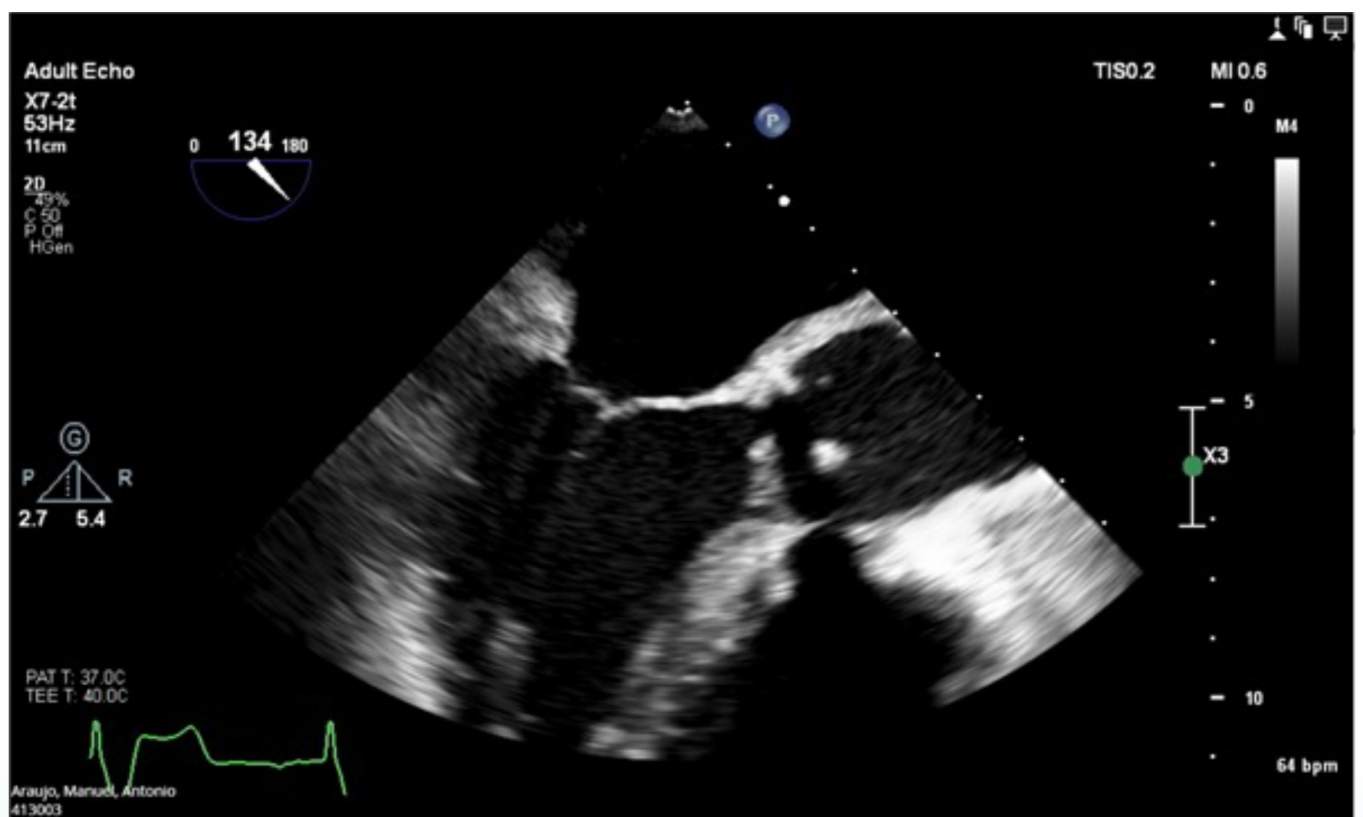
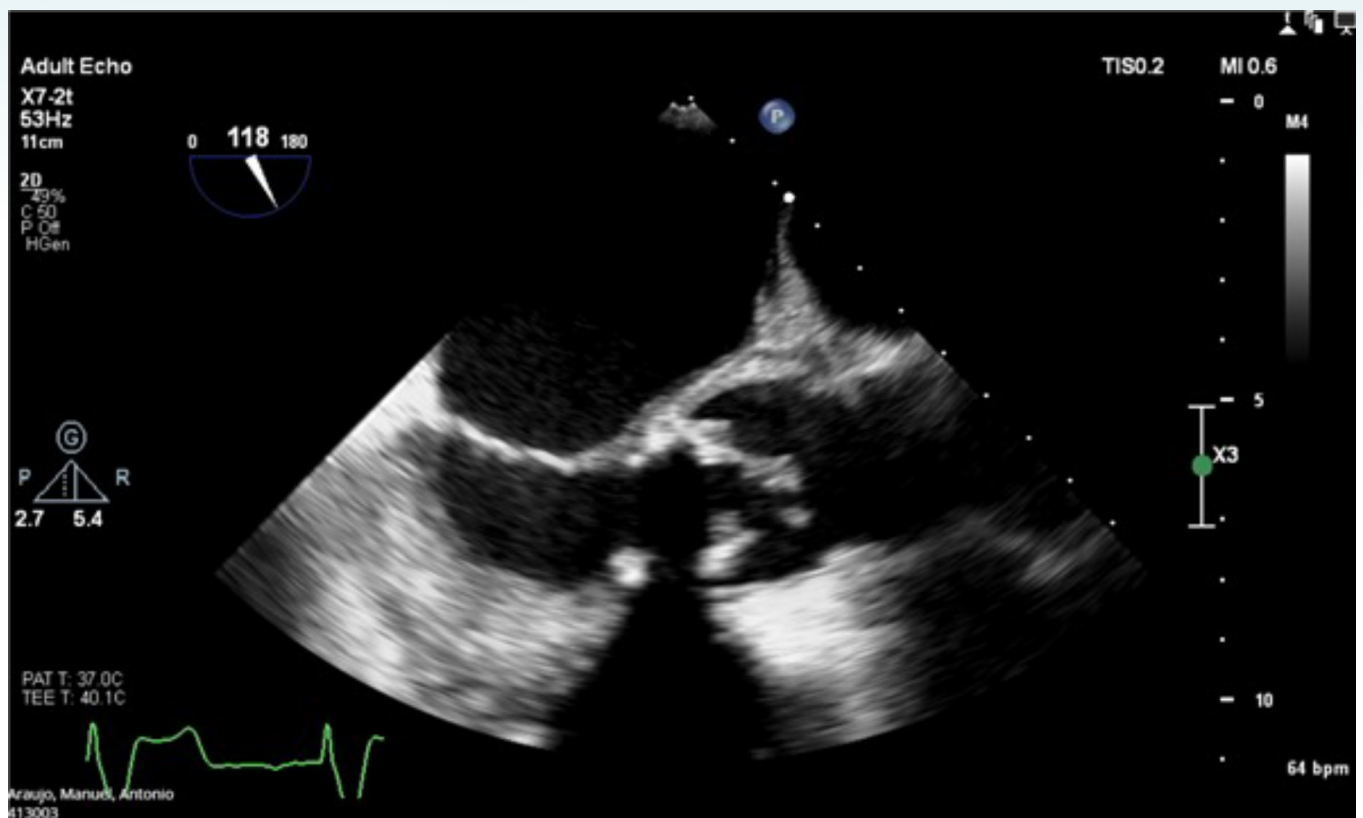
Three days after clinical stabilization, the patient presented high fever with shivering. Central venous catheter (CVC) was removed for suspected infection, and empiric treatment was started with vancomycin and ceftriaxone. Fever and persistently high inflammatory parameters led to a chest and abdominal CT scan to exclude other possible foci of infection. Splenic infarcts were observed suggesting embolic phenomena; CVC culture was negative and *Propionibacterium acnes* was identified in one set of blood cultures after 9 days.

Considering a possible EI, a transesophageal echocardiogram was performed, revealing a 10x5mm vegetation in the aortic bioprosthesis, confirming the diagnosis of prosthetic aortic valve endocarditis, with no evidence of periannular complications (fig. 1 and 2).

DISCUSSION

EI caused by *Propionibacterium acnes* is rare (~0.3% of all cases), but have been increasingly described in the literature, especially in association with prosthetic heart valves. The diagnosis is difficult since clinical presentation is usually indolent. Additionally, blood cultures may be negative when incubated for only 5 days. In this case, the incubation period was extended, allowing the identification of *P. acnes* after 9 days, otherwise this might not have happened, and the diagnosis missed. Therefore, it is common that patients present with advanced disease, such as embolic complications. Here, we hypothesize that embolic stroke may have been the first manifestation of the disease and acute myocardial infarction was another form of arterial embolization, explaining why embolic events occurred despite proper anticoagulation.

The indolent nature of the infection leading to the delayed diagnosis might explain the high incidence of complications and advanced disease in *P. acnes* IE. Extended incubation of blood cultures and multimodality imaging may be the key to early diagnosis in the presence of clinical suspicion.



CC 38

MINOCA Unveiled: Pheochromocytoma as an Uncommon Etiology of Myocardial Infarction with Non-Obstructive Coronary Arteries

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INTRODUCTION

Pheochromocytoma is a rare catecholamine-producing neuroendocrine tumor of the adrenal gland which typically presents as episodic palpitations, hypertension, and sweating. However, some patients may exhibit symptoms resembling cardiac emergencies, including arrhythmias, ST and non-ST elevation myocardial infarction and heart failure.

CASE PRESENTATION

A 45-year-old female patient presented to the emergency department due to sudden-onset epigastric pain radiating to the precordial area and complaining of previous episodes of palpitations, sweating and headache over the past 3 months. Medical history included Graves' disease with normal thyroid function and recently diagnosed arterial hypertension treated with azilsartan 40 mg daily. On admission, she was tachycardic with a blood pressure of 98/67mmHg. The admission ECG (with pain) revealed a J point and ST elevation <1mm in DI-aVL and 1 mm ST depression in inferior leads; alterations that were transient and evolved with T-wave inversion in DI-aVL and biphasic T-wave in V2. Blood tests showed elevated high-sensitivity troponin (3359 pg/ml) and D-dimer. Chest-CT angiogram excluded pulmonary embolism but incidentally revealed a 47mm solid mass in the left adrenal gland. Transthoracic echocardiogram was unremarkable. She was diagnosed with nonST elevation myocardial infarction, admitted to the intensive care cardiac unit where she stayed for 4 days remaining clinically stable and with-

out pain relapse. Coronary angiogram ruled out obstructive coronary artery disease. Specific analysis for adrenal tumors revealed elevated urinary metanephrines and plasmatic catecholamines. Abdominal CT-scan showed a 47x46mm left adrenal tumor with a mean attenuation coefficient of 42 Hounsfield units, suggesting pheochromocytoma. The final diagnosis was myocardial infarction with non-obstructive coronary arteries (MINOCA) possibly caused by episodic vasospasm resulting from the untreated pheochromocytoma or, less likely, due to catecholamine-induced cardiomyopathy. She was referred for surgery and awaits adrenalectomy.

DISCUSSION

Pheochromocytomas may present with potentially fatal cardiovascular complications resulting from the potent effects of the catecholamine surge. Most patients have normal left ventricular ejection fraction on TTE, although around 10% may develop catecholamine-induced cardiomyopathy. Proposed mechanisms include microvascular dysfunction, multivessel epicardial spasm and catecholamine-mediated myocardial injury. Treatment with fenoxibenzamine followed by surgical intervention typically results in the reversal of cardiac manifestations. This case highlights pheochromocytoma as an unusual cause of MINOCA, which was suspected due to incidental finding of an adrenal tumor, further supported by the patients' preceding symptoms and confirmatory laboratory tests.

CC 39

Clearing the Way: Pulmonary Endarterectomy in Chronic Thromboembolic Pulmonary Hypertension

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INTRODUCTION

Chronic thromboembolic pulmonary hypertension (CTEPH) is an uncommon and underdiagnosed complication of acute pulmonary embolism, whose true prevalence is still unclear. CTEPH is characterized by intraluminal thrombus organization, resulting in progressive right heart failure.

CASE PRESENTATION

A 27-year-old smoker woman, with a background of obesity and suspected hypertension (with prior exclusion of secondary causes), presented in emergency department after an episode of syncope. Her home medications included aspirin, bisoprolol and a combined oral contraceptive pill. On clinical examination, the patient appeared anxious, hypotensive and tachycardic. Arterial blood gas analysis showed respiratory alkalosis with type 1 respiratory failure. Laboratory tests revealed a positive plasma D-dimer result, NT-proBNP 9260 pg/mL and normal troponin I. The electrocardiogram showed sinus tachycardia with a S1Q3T3 pattern. It was decided to proceed with computed tomography pulmonary angiography (CTPA), which showed bilateral pulmonary thromboembolism involving both pulmonary arteries and their segmental and subsegmental branches. Treatment was initiated with enoxaparin. Doppler ultrasound showed signs of recent deep vein thrombosis in the left lower limb. Transthoracic echocardiography showed signs of pulmonary hypertension, with an estimated systolic pulmonary artery pressure (sPAP) of 110 mmHg. The

thrombophilia workup revealed heterozygosity for MTHFR C677T, MTHFR A1298C, and factor V H1299R. The estimated sPAP was gradually reduced to 92 mmHg and a subsequent CTPA showed substantial reduction in signs of bilateral thromboembolism. The patient remained hemodynamically stable during hospital stay and was discharged home on the 21st day medicated with spirinolactone, furosemide, ramipril and warfarin. A CTPA was performed 3 months after discharge, revealing chronic bilateral pulmonary thromboembolism. Right heart catheterization showed signs of pre-capillary pulmonary hypertension. During follow-up the patient complained of fatigue, with a World Health Organization functional Class III, even after starting phosphodiesterase-5 inhibitor sildenafil. A pulmonary thromboendarterectomy was performed approximately 1 year after discharge, with subsequent suspension of sildenafil. Post procedure echocardiogram showed a clear improvement in the signs of pulmonary hypertension, with an estimated sPAP of 26 mmHg. As of today (5 years after diagnosis) the patient remains in stable clinical condition, with a World Health Organization functional Class I.

DISCUSSION

Treatment of CTEPH often requires a multidisciplinary approach and may involve surgery, medical treatment, or both. CTEPH patients should receive lifelong anticoagulation, whereas the treatment of choice for patients who remain symptomatic is pulmonary endarterectomy.



CC 40

A less obvious cause of pericardial effusion

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INTRODUCTION

Anorexia nervosa is associated with numerous medical complications that are directly attributable to weight loss and malnutrition. The complications affect most major organ systems and account for approximately half of all deaths in anorexia nervosa.

In the cardiovascular system, anorexia nervosa can cause structural and functional abnormalities. These may include decreased cardiac mass, myocardial fibrosis, pericardial effusion, bradycardia, hypotension, or higher risk for arrhythmias.

Pericardial effusion has been reported in patients with anorexia nervosa, but the prevalence and etiology are not clear. Some prospective studies report prevalence around 30% with resolution in about 60-80% of cases after refeeding.

CLINICAL CASE

We present the case of a 20-year-old woman admitted to the Internal Medicine department in the context of anorexia nervosa with a restrictive pattern, leading to severe malnutrition and cachexia.

Her medical history includes childhood obesity and a history of bullying. In 2020, when she weighed 100 kg, she started a progressively more restrictive diet and daily exercise routine, resulting in a weight loss of 40 kg over 2 years. She reports that she lost control over her eating habits, and despite recognizing that she had reached a healthy weight, she couldn't return to a normal diet.

On admission, the patient presented with 38Kg, BMI 13.15 kg/m² and symptoms of asthenia, significant pe-

ripheral edema, petechial rash on the lower limbs, precordialgia, dyspnea on moderate effort, bradycardia, and had been in amenorrhea for 3 months.

The complementary study showed thrombocytopenia, acute kidney injury, marked hepatic cytolysis, hyponatremia, and a moderate volume pericardial effusion (29mm).

The pericardial effusion was limited to the right heart chambers and remained globally overlapping throughout the hospitalization, without hemodynamic repercussion and with no indication for therapeutic pericardiocentesis.

Given the overall clinical presentation and after excluding other differential diagnoses such as infectious or autoimmune diseases, it was considered that severely restrictive anorexia nervosa would explain the whole spectrum of medical complications observed.

Seven months after discharge, the patient is now at an appropriate weight, with normalization of analytical changes and resolution of the pericardial effusion.

DISCUSSION

The importance of this case lies in the fact that it represents an uncommon cause of pericardial effusion. It is important to raise awareness of the cardiovascular complications associated with severe cases of Anorexia Nervosa.

It should be emphasized that, similar to what happened with our patient, pericardial effusions are typically clinically silent, without signs of cardiac tamponade, and generally regress with weight restoration.

CC 41

In the presence of a silent fever, endocarditis speaks louder

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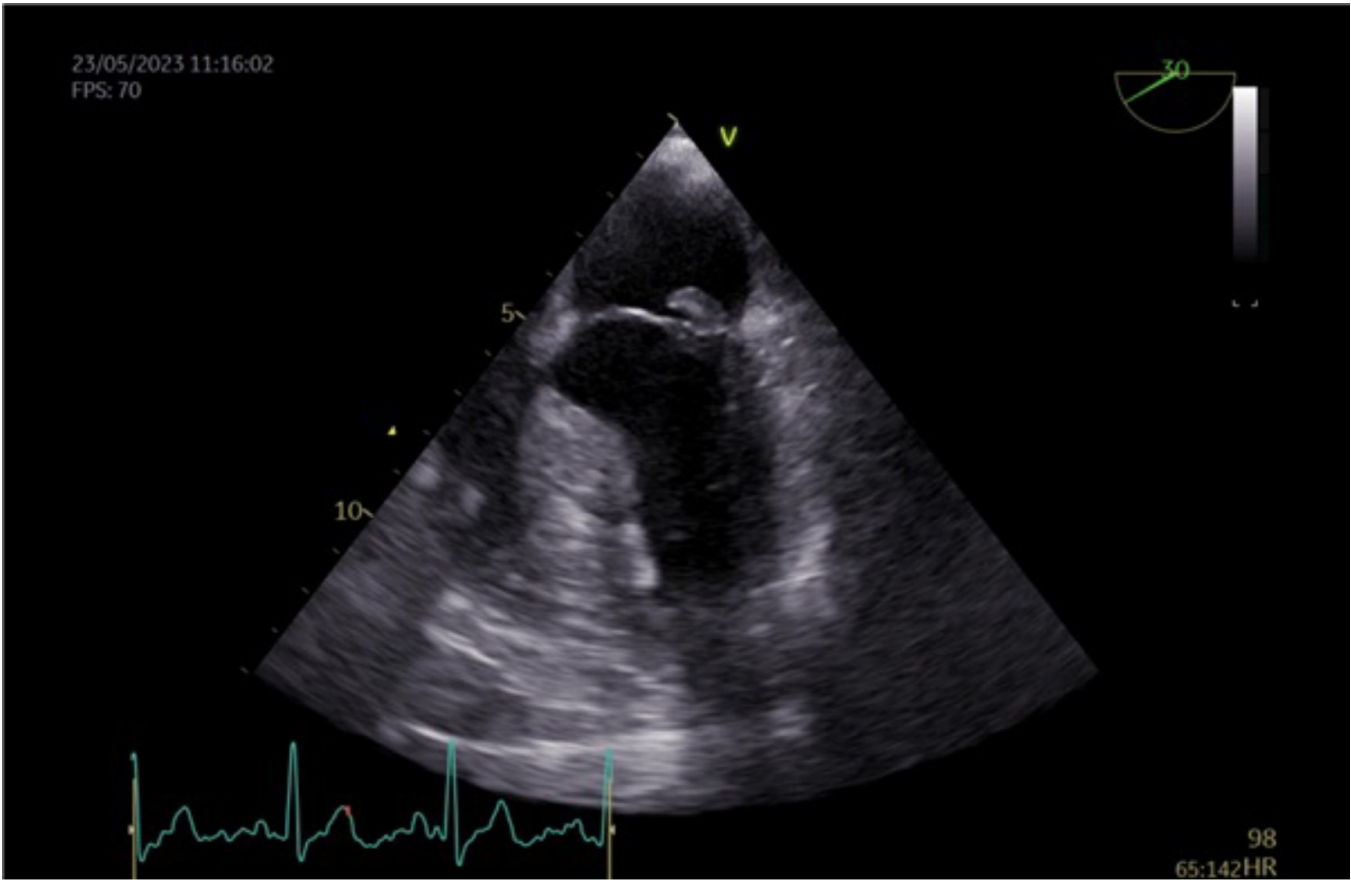
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We describe a case of a patient admitted to the emergency department with prolonged fever and intolerance to exertion. The lack of a distinct infectious focus, combined with the detection of a systolic murmur and previous knowledge of valvular structural changes, prompted the consideration of a challenging diagnosis, that was confirmed with blood cultures and transesophageal echocardiography.

A 69-year-old male was admitted to the emergency department with a 2-day history of fever and fatigue, following a course of antibiotics. The fever began 4 weeks earlier, with no discernible focus of infection and was accompanied by intolerance to exertion. The patient's fever, peaking at 39°C, responded well to paracetamol. Its occurrence was not associated with any specific time of day. This ailment prompted the patient to visit the emergency department on three separate occasions, during which three courses of antibiotics were administered. The fever subsided after each treatment but re-emerged within two days of its completion. Upon admission, the patient's hemodynamics were stable, but tachycardia (BP 110/67 mmHg, HR 117 bpm) and hyperthermia (TT 38°C) were observed. Upon physical examination, the patient presented with slightly discolored mucous membranes. Auscultation of the heart revealed rhythmic and regular S1 and S2, and the presence of a II/VI systolic murmur located in the mitral valve area without radiation. The rest of the examina-

tion was normal. The patient's medical history included treated malaria several years ago and a thickened mitral valve with a myxomatous appearance and prolapse of the posterior leaflet, identified in a previous echocardiogram. An analytical study revealed the presence of anemia, and elevated inflammatory markers. To investigate further, blood and urine cultures were taken, and the patient was started on antibiotics and hospitalized with a diagnosis of fever of unknown origin. While hospitalized, the hypothesis of native valve endocarditis was considered, and a transesophageal echocardiogram was performed. It revealed the presence of a mass adhering to the auricular surface of the posterior leaflet, measuring 5x7mm at 0° and 9x6mm at 120°. No evidence of infectious involvement of the mitral annulus was found. Blood cultures identified *Streptococcus agalactiae*, which was found to be sensitive to penicillin. The patient was then started on this medication, leading to an improvement in his condition.

This is an atypical manifestation of endocarditis caused by a rare pathogen. Typically, Group B *Streptococci* endocarditis is characterized by acute onset and is highly destructive, often resulting in embolic events. However, this was not observed. The purpose of this exposition is to emphasize the importance of considering endocarditis as a potential diagnosis in cases of fever of unknown origin, especially in patients with pre-existing valvular abnormalities.



CC 42

“Unblocking” Fabry disease: an atypical presentation

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Fabry disease (FD) is a rare X-linked inherited lysosomal storage disorder, due to a mutation in the *GLA* gene resulting in lower α -galactosidase A (α -Gal A) enzyme activity. It results in the accumulation of globotriasylceramide in affected tissues, namely the heart, kidneys, vasculature and peripheral nervous system. Cardiac manifestations of FD include left ventricular hypertrophy (LVH) and fibrosis, valve thickening or regurgitation, heart failure, angina, dysrhythmias, cardiac conduction abnormalities and sudden death. Despite available therapies and supportive treatment, cardiac involvement carries a major prognostic impact, representing the main cause of death in FD.

We report the case of a 64-year-old man admitted to the emergency room for syncope that resulted in head injury. He had no relevant clinical history and no regular medication. The patient mentioned sudden cardiac death of his mother at age 60. Electrocardiogram (ECG) revealed a third-degree atrioventricular block with 25bpm ventricular escape rhythm. Initial blood tests showed no metabolic or ionic disturbance and head tomography was normal. Emergency temporary transvenous right ventricular pacing electrode was placed as bridge to a definitive dual chamber device. The patient was discharged for outpatient follow-up. After 6 months, pacemaker interrogation showed only 1.5% of p-synchronous pacing. ECG revealed left bundle branch block (LBBB) and echocar-

diography documented left ventricular ejection fraction of 50%, abnormal movement of the septum in relation with LBBB, without hypertrophy or valve changes. Magnetic resonance imaging showed a LVEF of 44% due to dyssynchrony, with no fibrosis or late gadolinium enhancement and normal native T1, without signs of ischemia. Serologies for Borrelia, viral hepatitis and HIV were negative. Serum and urinary protein electrophoresis were normal, as well as levels of angiotensin converting enzyme. Genetic testing for mutations in the *TTR* gene showed no pathogenic variants, but it was found a probably pathogenic mutation (c.65T>C) in the *GLA* gene. At follow-up, the patient remained asymptomatic, without signs of heart failure, specific neurologic signs or classic skin findings of FD. The patient was then referred for further genetic counselling, screening of first-degree relatives and metabolic disease consultation.

This case highlights an atypical presentation of cardiac FD. Although the hallmark of cardiac involvement in FD is LVH, this patient presented only with a severe conduction defect without other suggestive clues. Indeed, cardiac arrhythmias are common in FD but are mostly present in subjects with LV structural abnormalities. As seen in our patient, FD would have been missed, if not looked for. So, one must be alert for unusual etiologies of AV block, keeping in mind the screening for FD in male patients with arrhythmia requiring a pacemaker.

CC 43

Acute coronary syndrome post-radiotherapy: a clinical case

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Radiotherapy (RT) is associated with ischemic heart disease (IHD) due to incidental cardiac radiation exposure. Thoracic radiation can damage virtually any component of the heart - myocardium, pericardium, valves, coronaries and conduction system. Although technological progress has decreased radiation doses delivered to normal tissues, significant heart doses still can't be completely avoided. Indeed, women irradiated for left-sided breast cancer (BC) receive substantially higher doses to the heart than women irradiated for right-sided BC. Several trials show an increased IHD risk after RT in left-sided BC, but acute coronary syndrome (ACS) is quite rare during left breast irradiation.

We report the case of a 51-year-old female with history of type 2 diabetes, dyslipidemia and asthma. She was diagnosed with a HER2-positive left-sided BC and submitted to neoadjuvant chemotherapy with doxorubicin, cyclophosphamide and paclitaxel, followed by a mastectomy with axillary lymph node dissection. She was then started on trastuzumab and adjuvant radiotherapy to the left chest wall and axilla (50Gy) with an additional "boost" on the scar (total dose 60Gy). During RT treatments, she presented with recurrent self-limited episodes of an oppressive precordial chest pain at rest, with no other symptoms. Radiation-induced pericarditis *versus* costochondritis was assumed and non-steroid anti-inflammatory drugs were prescribed with

partial relief. One month later she performed an echocardiogram revealing preserved systolic function, with hypokinesia of the basal segment of the inferior wall, previously unknown. The electrocardiogram showed a *de-novo* T-wave inversion in aVL lead. Cardiac troponins on routine evaluation were negative. She was afterwards evaluated in Cardio-Oncology clinic and performed further study with: cardiac magnetic resonance that confirmed hypokinesia localized to a small basolateral area and the medio-apical inferolateral wall, associated with minimal late subendocardial enhancement with coincident perfusion defect; and also, coronary computed tomography, documenting a stenosis of 70-99% in the distal right coronary artery. She was started on bisoprolol, aspirin, atorvastatin and amlodipine, remaining asymptomatic under optimal medical therapy.

This case illustrates an example of IHD in a patient under RT, peculiar for its presentation as an ACS. It is known that radiation leads to an increase of myofibroblasts and macrophages, producing intimal proliferation and a pro-thrombotic condition. However, RT damages remain clinically silent for a long period after RT, therefore ACS is unlikely to occur during treatment. This case highlights the importance of baseline cardio-oncology risk stratification and optimization of prevention strategies in order to minimize the cardiac side effects of RT and ensure effective oncological treatments.

CC 44

An unusual case of inappropriate ICD activation - the Reel Syndrome

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INTRODUCTION

Although rarely seen in clinical practice, lead dislodgement (LD) is an important cause of cardiac implantable electronic devices (CIED) malfunction which may present as three different syndromes (Ratchet, Reel and Twiddler). These entities should be considered when patients report implantable cardioverter-defibrillator (ICD) shocks or neuromuscular symptoms (mostly diaphragmatic or brachial), and when device interrogation shows inappropriate therapies or parameters.

CASE DESCRIPTION

A 70-year-old man with a history of non-ischemic dilated cardiomyopathy and severe left ventricular dysfunction, despite optimized medical therapy, underwent implantation of a single-chamber ICD at our hospital for primary prevention of sudden cardiac death. A transvenous active fixation electrode was placed at the lower septum of the right ventricle (RV). The postprocedural period was uneventful and adequate positioning and programming parameters were confirmed before hospital discharge.

On the 40th post-implantation day, a home monitoring shock delivery alert was received. Device interrogation showed a noise episode associated with an increase in lead impedance assumed as ventricular fibrillation, which led to an inappropriate therapy. The patient confirmed having felt the shock without other cardiac symptoms. A chest X-ray was performed and showed dislodgement and coiling of the lead around the generator, back to the

pocket, without torsion. When asked, the patient admitted external manipulation of the device due to pocket discomfort. Diagnosis of LD as Reel syndrome was made.

Consequently, the patient was admitted for rhythm monitoring and ICD lead replacement, as therapies had to be turned-off. The lead was removed uneventfully and a new transvenous electrode was implanted in the RV apex and connected to the same ICD device without complications. Hospital discharge occurred on the following day, after confirmation of adequate positioning and programming parameters. At the 6-month scheduled visit, normal ICD functioning was confirmed without any new events.

DISCUSSION

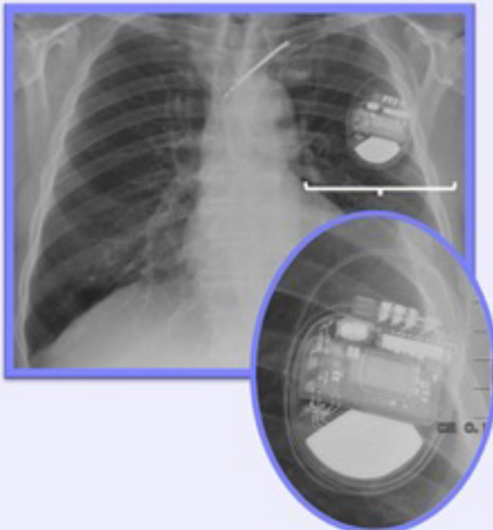
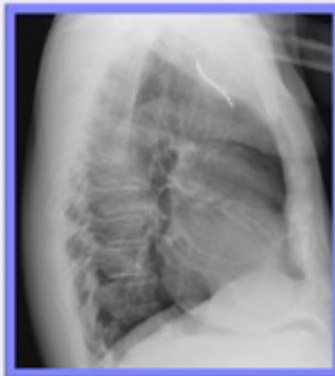
We present a case of inappropriate shock delivery that brought up an underlying ICD dysfunction due to LD. Device interrogation and X-ray made the final diagnosis of Reel syndrome, a rare but important complication of CIED insertion characterized by the device's rotation on its transverse axis with subsequent lead retraction and coiling around the generator, without any lead damage or twisting. Typically happening within the first months after insertion as in this case, it is associated with some risk factors, here advanced age and external manipulation. Early intervention with lead replacement is crucial for preventing potential life-threatening events. Home monitoring was key to early detection, as a late diagnosis can lead to repetitive inappropriate shocks due to lead noise.

Figure 1: Importance of X-Ray in the diagnosis of lead dislodgement syndromes.

After ICD implantation:
RV lead well positioned.



Reel Syndrome:
Note lead coiling around the ICD pulse generator.



ICD: implantable cardioverter-defibrillator; RV: right ventricle.

CC 45

A pertinent fast revascularization in non-ST-segment elevation acute coronary syndrome

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INTRODUCTION

Immediate coronary angiography in the setting of non-ST elevation acute coronary syndrome is recommended in a specific subset of patients, to be managed as ST elevation myocardial infarction. These include patients with recurrent pain, hemodynamic instability, acute heart failure, life-threatening arrhythmias, among others. Identifying these patients is not always straightforward.

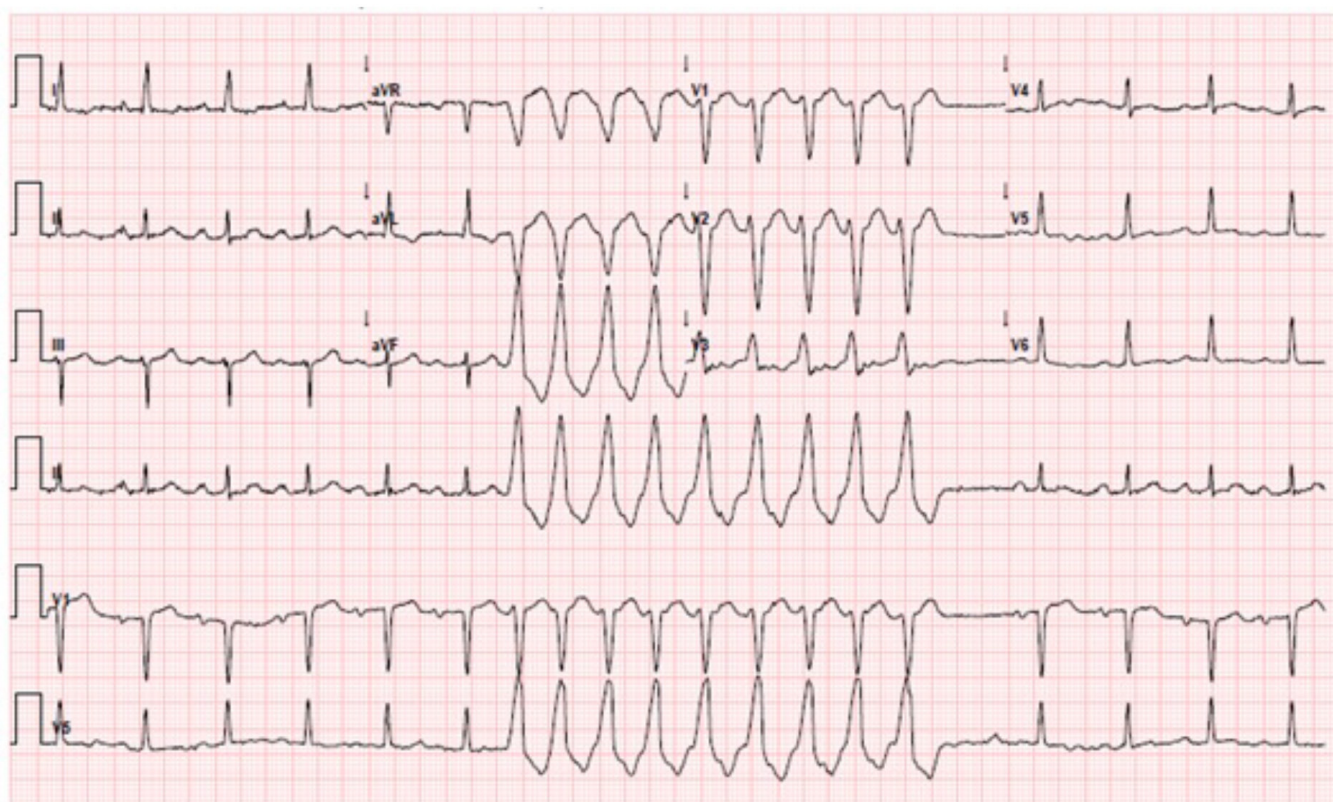
CLINICAL CASE

70 years-old male, past medical history of arterial hypertension, type 2 diabetes, obesity and hypothyroidism, presented to the emergency department with complaints of intermittent anterior thoracic pain, with clear angina characteristics, for the last 5 days. Patient also mentioned fatting spells in the previous evening, with no syncope. He was conscious, hemodynamically stable, with peripheral edema, rales in pulmonary auscultation and hypoxemia. Electrocardiographic monitoring showed runs of monomorphic ventricular tachycardia (VT), 7 to 10 complexes, continuously alternating with sinus rhythm with no specific changes suggestive of ischemia in the 12 lead ECG. Markers of myocardial necrosis were normal. Transthoracic echocardiogram showed non-dilated left ventricle, hypokinesia in the medio-apical seg-

ments of the anterior wall and apical aneurysm as well as moderately depressed systolic function. He was started on amiodarone and transported to a tertiary center. Invasive coronariography demonstrated left anterior artery (LAA) with chronic occlusion and posterolateral artery with 90% stenosis; 1 drug-eluting stent was placed in this vessel. The runs of VT ceased after angioplasty, with only rare premature ventricular beats, as well as the thoracic pain.

DISCUSSION

The chronic occlusion of LAA was indicative of scar tissue in its territory, with the possibility of VT arising from here, contributing to thoracic pain due to coronary hypoperfusion. The culprit posterolateral artery could also be producing this form of ventricular arrhythmia, explaining its ending upon performing angioplasty. Regardless of the causality, opening the vessel was determinant to stop the VT, as well as preventing possible evolution to cardiogenic shock. This subset of high-risk patients can be considered in the *ongoing myocardial ischemia* category and should be rapidly identified and treated as swiftly as patients with ST elevation myocardial infarction.



CC 46

The other face of an unsuspected mass

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INTRODUCTION

The lipomatous hypertrophy of the interatrial septum (LHIS) is a common benign finding in the transthoracic echocardiogram. Nevertheless, some visual aspects, such as large dimensions or unusual contour, might raise the suspicion of a different etiology.

CLINICAL CASE

Male, 57 years-old, admitted for acute ST-elevation myocardial infarction, with significant stenosis of the left anterior descending artery, submitted to primary angioplasty with placement of 1 drug-eluting stent. Left ventricular ejection fraction (LVEF) of 47%, with hypokinesia of the antero-septal territory. Previous medical history included type 2 diabetes *mellitus*, arterial hypertension, obesity and hypercholesterolemia. He was medicated with dual antiplatelet therapy, betablocker, high-intensity statin, neurohormonal antagonists and insulin. After treatment, the patient kept asymptomatic, with no disturbances of rhythm or conduction.

Follow-up echocardiogram showed non-dilated left ventricle, akinesia of mid-apical segments of the inferior

interventricular septum and anterior wall; LVEF 40%, global longitudinal strain -10%. A mass in the upper part of the right atrium was also noted, with maximal dimensions of 2.8 x 2.2 cm.

DISCUSSION

Despite its benign nature, LHIS can be associated with increased risk of arrhythmia, by conduction disturbance or sinoatrial node disease, so clinical surveillance should be kept close. The transesophageal echocardiogram is the most useful exam for the patient as it allows a good delimitation of the mass and it excludes intracavitary thrombus. The computed tomography also enables three-dimensional reconstruction of this sort of masses, but it does not have the same value to exclude thrombus. The cardiac magnetic resonance is an imaging method with high specificity for adipose tissue, so it carries great advantage for these situations as it can be used for differential diagnosis in a non-invasive way. These imaging modalities exempt the patients from endomyocardial biopsy and unnecessary surgery, in the absence of symptoms.

CC 47

The mass that looks like a marble

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INTRODUCTION

An intracardiac mass found in a transthoracic echocardiogram always requires a differential diagnosis in order to appropriately guide the patient.

CLINICAL CASE

73 years-old female patient, previous medical history of type 2 diabetes, obesity and arterial hypertension, presented to the emergency department with fatigue and dyspnea. She had a complete AV block, probably due to hiperkaliemia (6.6 mEq) in the context of renal failure and metabolic acidosis. She was conscious and hemodynamically stable but with respiratory failure secondary to heart failure, with need of non-invasive ventilation. The AV block disappeared after kaliemia correction. Transthoracic echocardiogram was requested to evaluate structural cardiopathy, which showed normal systolic function, slight concentric hypertrophy, calcified mitral valve and a pediculated mass (dimensions 17 mm x 5.8 mm) next to the subvalvular apparatus of the anterior

mitral leaflet. The characteristics suggested a fibroelastoma. A trial of anticoagulation showed no changes in the mass. Hemocultures were negative and no embolic event was found in cerebral and thoraco-abdominal computed tomography.

DISCUSSION

Benign masses found in echocardiogram are not uncommon, both intracardiac and, much less frequently, extracavitary. There can be several consequences for the patients, such as systemic embolization, so usually surgical excision is recommended. Fibroelastomas are very mobile, usually found in the valvular leaflets, with very well-defined contours - one could say they look like a glass marble rolling around. They can be associated with increased endocardial trauma after surgery, radiation or in the context of hypertrophic myocardiopathy. The final diagnosis is made upon histological analysis after excision.

CC 48

Exercise-Induced Atrioventricular Block and Hypertrophic Cardiomyopathy: Can Right Ventricular Pacing be Just Right? - A Case Report

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INTRODUCTION

Sudden cardiac death is one of the most devastating complications of hypertrophic cardiomyopathy (HCM). Sudden death is usually a consequence of ventricular arrhythmias, however, it can also be secondary to cardiac conduction disturbances. We report a symptomatic 67-year-old woman with exercise-induced atrioventricular (AV) block and HCM successfully treated with right ventricular pacing.

CASE REPORT

We present a 67-year-old woman who sought a cardiology consultation due to breathlessness and dizziness associated with medium-to-high-intensity efforts. Comorbidities were arterial hypertension and dyslipidemia. No relevant family history. Auscultation revealed a systolic murmur heard at the upper right sternal border.

Transthoracic echocardiography (TTE) revealed flow acceleration in the left ventricular outflow tract (LVOT) without significant intraventricular gradients (IVG) and mild mitral regurgitation (MR). A 24-hour Holter revealed day-time Mobitz II 2:1 second-degree AV block, which occurred during periods of higher physical exertion and coincided with the patient's symptoms. Resting ECG showed 1:1 atrioventricular conduction. Stress testing confirmed this finding.

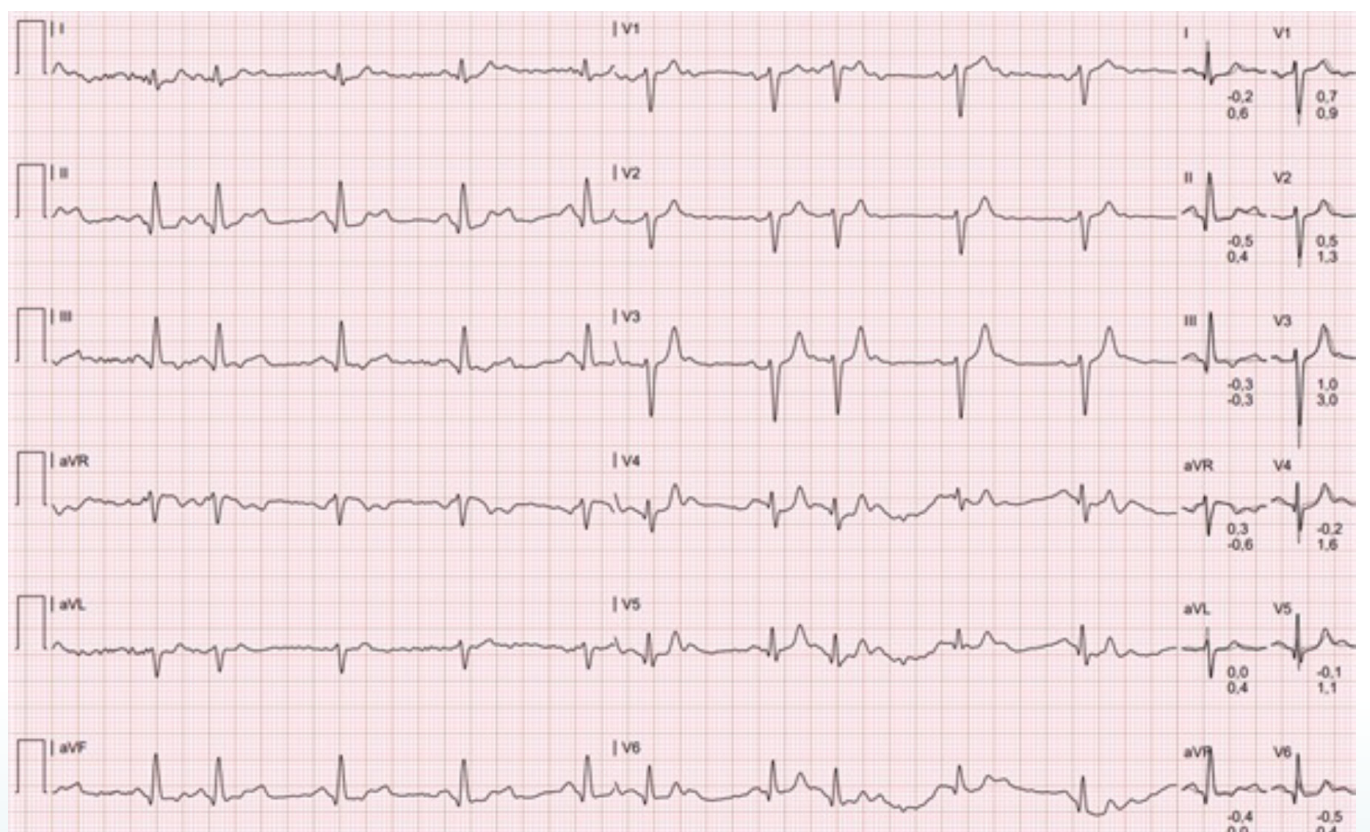
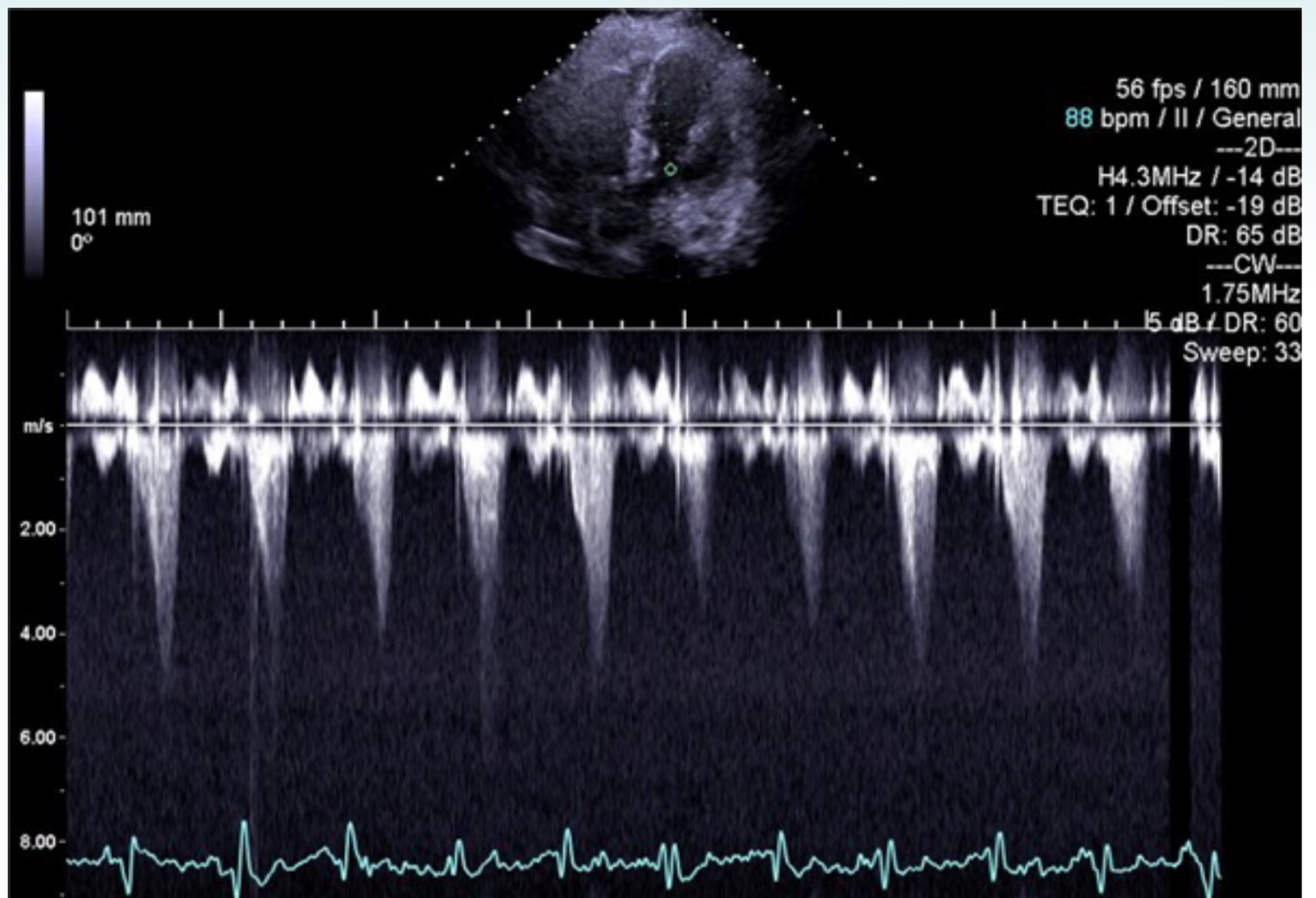
A coronary computed tomography angiography excluded obstructive epicardial coronary artery disease. Finally, a cardiac magnetic resonance demonstrated features consistent with HCM, mainly, an interventricular sep-

tum measuring 17 mm, systolic anterior motion of the mitral valve at rest, and a heterogeneous pattern of late gadolinium enhancement. Exercise-induced AV block associated with obstructive HCM was assumed. The patient did not fulfill criteria for an implantable cardioverter defibrillator and, therefore, underwent implantation of a permanent dual-chamber pacemaker. However, she maintained effort-induced breathlessness.

An exercise stress echocardiogram (ESE) was therefore performed. In the beginning, while in the upright position, the IVG was already 60 mmHg. At peak effort, she developed an IVG higher than 100 mmHg and severe MR associated with worsening breathlessness. At this moment, AV conduction was interrupted with subsequent right ventricular pacing. Consequently, the IVG disappeared completely, as did her symptoms. In a multidisciplinary discussion, it was decided to reprogram the pacemaker to increase the right ventricular pacing percentage. A few weeks later, the ESE was repeated, revealing higher functional capacity and the absence of IVG.

DISCUSSION AND CONCLUSION:

Surgical myectomy and alcohol septal ablation (ASA) have become the main standards of therapy for symptomatic HCM patients with LVOT obstruction. However, the role of dual chamber pacing remains appropriate as the treatment strategy of choice in a subset of patients who refuse or are considered high risk for surgery and who are not suitable for ASA.



CC 49

Embolic stroke and misdiagnosed *Candida* endocarditis

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INTRODUCTION

Candida endocarditis (CE) is a rare and serious form of endocarditis that requires a high level of suspicion for timely diagnosis and appropriate treatment. The clinical presentation is highly variable and may include signs and symptoms of embolization.

CLINICAL CASE

A 73-year-old woman with a history of bioprosthetic aortic valve presented to the emergency department with aphasia and dysarthria that had started in the previous day. Cranioencephalic computed tomography revealed a small hypodensity in the left insula, indicating a subacute stroke. The patient was diagnosed with a partial anterior circulation infarct and was admitted to the stroke unit. The patient developed a sustained fever from the first day of hospitalization. Preliminary blood culture results showed the presence of yeast cells, leading to empirical therapy with fluconazole. Transoesophageal echocardiogram (TEE) revealed a mobile mass adherent to the right coronary cusp indicative of vegetation. Subsequently, the blood cultures grew *Candida* (*C.*) *famata*, leading to a switch in antifungal therapy to liposomal amphotericin B (AmB). The patient was transferred to the cardiology ward, and surgical intervention was postponed. A follow-up TEE revealed increasing of the size of the mass. Moreover, the patient experienced an episode of chest pain with elevated myocardial necrosis markers, suggesting a probable embolic event. Surgical

treatment was proposed and a new replacement with a biological aortic prosthesis was performed. Although, the microorganism isolated in the surgical specimen was *C. guilliermondii*. There were no complications, and the patient was discharged with fluconazole medication for one month.

DISCUSSION

Severe embolic complications may be the first and only indicators of fungal endocarditis, with cerebral embolization being the most commonly observed. Misidentification of *Candida* spp. is not uncommon, since some species share biochemical and morphological similarities. The use of molecular and proteomic methods for fungal identification has been recommended. Tailored treatment should be based on antifungal susceptibility testing and species identification. *C. guilliermondii* has a similar resistance profile to *C. famata*, there was no significant impact on the choice of initial treatment. However, the misdiagnosis can lead to an incorrect interpretation of antifungal susceptibility and inappropriate selection of the antifungal. Surgical timing should be carefully considered, and patient-specific factors play a crucial role. In conclusion, increasing awareness, early diagnosis, and implementing a comprehensive treatment strategy are crucial for improving the prognosis of CE and reducing its devastating impact on patients.

CC 50

Unraveling TTN Gene Mutation in Early-Onset Cardiomyopathy

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INTRODUCTION

The TTN gene is a gene that provides information for the production of titin, a protein located in the sarcomere of the striated muscle tissues. It limits the range of motion of the sarcomere and it is the third most abundant protein in muscle. Mutations in the TTN gene have been implicated as potential causes of hereditary myopathies characterized by early-onset cardiomyopathy.

CLINICAL CASE

A man at the age of fifty-five years old with multiple cardiovascular risk factors (including hypertension, dyslipidemi, and a history of smoking) and positive family history for sudden cardiac death was submitted to a definitive pacemaker due to a paroxysmal advanced atrioventricular block. An upgrade to cardiac resynchronization therapy pacemaker took place four years later due to asynchrony of the pacemaker and dysfunction of the left ventricle. Subsequently, the patient recovered the ejection fraction of the left ventricle to the normal range.

Considering the patient's pathology and age group, cardiac magnetic resonance imaging was performed, revealing a left ventricle size at the upper limit of normal/slightly dilated, severe depression of left ventricular global systolic function, global hypocontractility and ventricular dyssynchrony/pacemaker-mediated depolarization. An adenosine stress test was conducted, which yielded negative results for myocardial ischemia.

Moreover, the patient underwent genetic testing utilizing a peripheral blood DNA sample and a next-generation sequencing technique. The genetic test identified a heterozygous pathogenic variant, c.56648-1G>A, in the TTN gene. Current evidence suggests the likely pathogenic nature of this variant, which is associated with hereditary cardiomyopathy. This variant is located at a canonical splicing site and disrupts RNA splicing, potentially leading to the formation of a non-functional protein. It is located within the A band of TTN. In this case, the atrioventricular block causing the cardiomyopathy was probably an early manifestation of the genetic disease.

DISCUSSION

The identification of atrioventricular block causing cardiomyopathies at an early age should raise suspicion of underlying genetic conditions, particularly mutations in the titin gene. The prompt diagnosis of these conditions may put these patients under close surveillance of the left ventricle dysfunction. These conditions not only affect cardiac function but also have implications for other striated muscle tissue like muscles and should lead to appropriate family counseling.

CC 51

An Unexpected Finding in an Adolescent Rowing Athlete with Angina Pectoris - A Case Report

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INTRODUCTION

The development of significant intraventricular gradients (IVG) during exercise is rare, usually occurring with left ventricular hypertrophy. Chest pain is common in children and is a frequent reason for referral to pediatric cardiologists. Despite the benign nature of most pediatric chest pain, extensive and costly cardiac evaluation is common in these patients. In a group of athletes of all ages, one-third with a clear association between symptoms and exercise have IVG. In the case presented here, we describe an adolescent rowing athlete with excruciating effort angina only during upright exercise which was replicated while performing an exercise stress echocardiography.

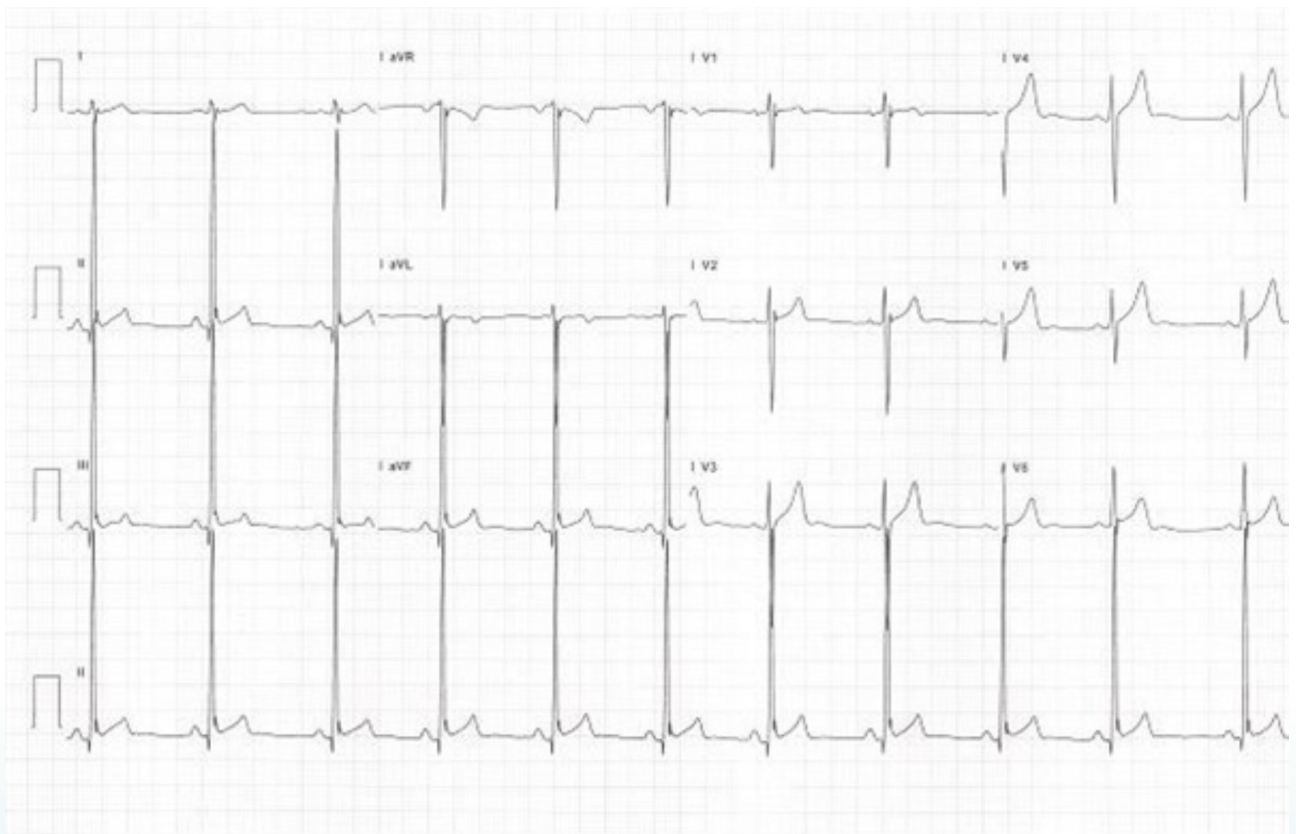
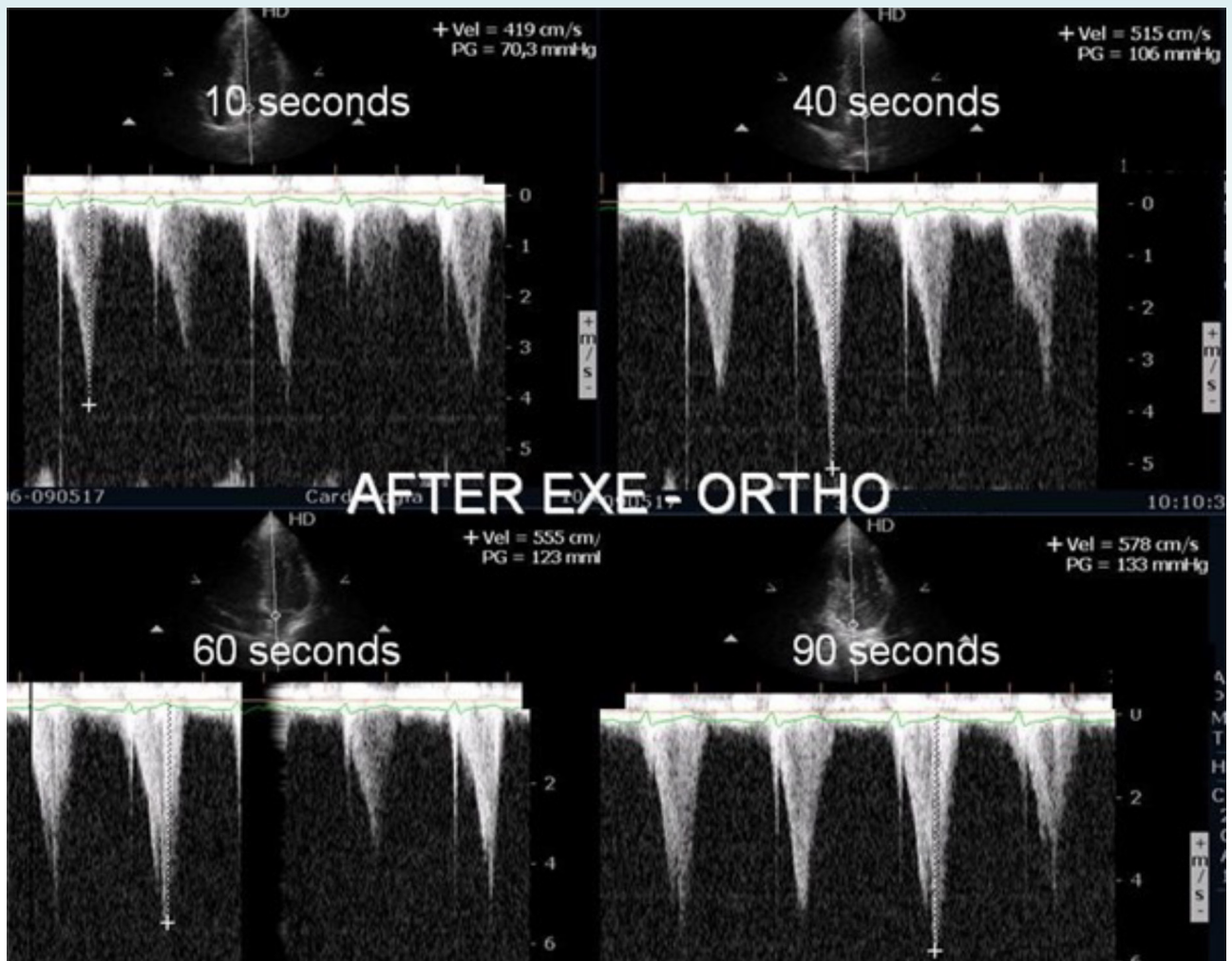
CASE REPORT

We describe the case of a 15-year-old Caucasian male, with a six-hour weekly training schedule, who complained of chest pain on and after strenuous running. He denied any symptoms while rowing. There was no relevant personal history, and no family history of sudden death or heart disease was reported. Physical examination revealed normal cardiac auscultation and normal radial, carotid, and femoral pulses. The twelve-lead electrocardiogram (ECG) was normal. The echocardiogram was also normal, with no left ventricular hypertrophy; the left ventricular end-diastolic diameter was 49 mm, the septum and posterior walls were 8 and 7 mm respectively, and the left ventricular outflow tract was 19 mm.

No abnormalities were found in the mitral valve and the sub-valvular apparatus. A treadmill exercise test was performed following the Bruce protocol that was considered positive for myocardial ischemia with ST-segment depression and chest discomfort that started 30 seconds after exercise. The patient underwent an exercise stress echocardiography (ESE), and immediately after the exam developed an IVG, which increased to 133 mmHg 90 seconds into the recovery period - a systolic murmur was noted at the left sternal border at that moment - and disappeared at 3 minutes. At this moment systolic anterior movement (SAM) of the mitral valve could be observed. The ESE was negative for myocardial ischemia, although the athlete developed severe chest discomfort and ST-segment depression on the ECG after exercise in the orthostatic position.

CONCLUSION AND DISCUSSION

The case described, in which significant abnormalities in cardiac function were found only immediately after exercise in the orthostatic position, highlights the essential role a normal venous return has in maintaining cardiac function. When physical activity is stopped abruptly while in the orthostatic position, a sudden decrease in venous return/preload occurs secondarily to diminished skeletal muscle contraction of the lower limbs (skeletal muscle pump). This leads to increased intraventricular gradients and exercise-related symptoms due to end-systolic obstruction secondary to ventricular cavity obliteration.



CC 52

2R: A rare cardiac mass with a rare location

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INTRODUCTION

Cardiac primary tumors are exceptionally rare. The specific signs and symptoms are dictated by the anatomical location but the diagnosis is frequently incidental during routine cardiac imaging. More than 75% of cardiac tumors are benign, the most common types in the adults being myxomas, fibroelastomas and lipomas, respectively.

CASE PRESENTATION

A 68-years-old female presents complaints of new-onset fatigue, dizziness and palpitations. Her past medical history was relevant for atrial tachycardia ablation in 2018 and a re-ablation 6 months before, dyslipidemia and former tobacco misuse. At physical exam she had an irregular pulse, so an EKG was performed and de-novo atrial fibrillation with controlled ventricular rate was shown. Anticoagulation was started with a DOAC. An echocardiogram (ECHO) was later performed in sinus rhythm and a mobile round 11.58 x 9.74 mm isoechogenic mass in the septal leaflet of the tricuspid valve was detected, conditioning mild tricuspid regurgitation. Biventricular function was normal. No other significant changes were found (Figure 1 - Transthoracic Echocardiogram). We suspected subacute endocarditis or a thrombus due to AF with pulmonary embolization. Blood analysis were within the range. Endocarditis and

pulmonary embolism were excluded. DOAC was changed for warfarin. An ECHO was repeated 15 days after and since the mass presented the same exact dimensions, a cardiac magnetic resonance imaging was conducted. A small mass was observed attached to the base of the auricular surface of the septal leaflet of the tricuspid valve, with tissue characteristics suggestive of papillary fibroelastoma (Figure 2 - Cardiac Magnetic Resonance Imaging). The patient was proposed for cardiac surgery.

DISCUSSION

Cardiac papillary fibroelastoma is more common in males between 60 and 80 years-old. It usually affects the valves of the left chambers and only up to 9% of cases involves the tricuspid valve. Clinical presentation ranges from absent symptoms (30%) to syncope/presyncope, fatigue, dyspnea, stroke, myocardial infarction, heart failure, sudden cardiac death, due to systemic or pulmonary embolic events. Large tumors (> 1 cm), those with high mobility or those responsible for any embolic event should undergo cardiac surgery. We report the case of a rare cardiac mass with a rare location conditioning symptoms of fatigue, dizziness and palpitations, therefore highlighting the importance of clinical suspicion of this pathology in clinical practice.

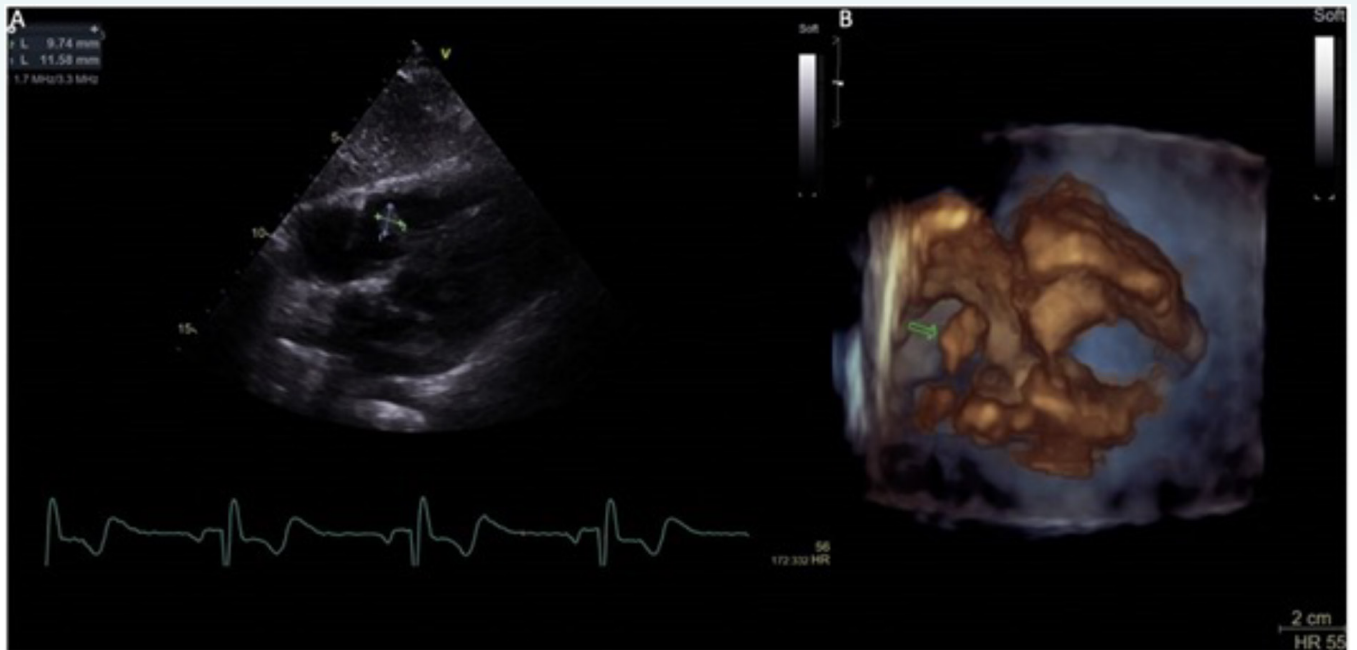


Figure 1 – Transthoracic echocardiogram. A – Subcostal view showing a mobile round isoechogenic mass in the septal leaflet of the tricuspid valve. B – 3D view of the aforementioned mass.

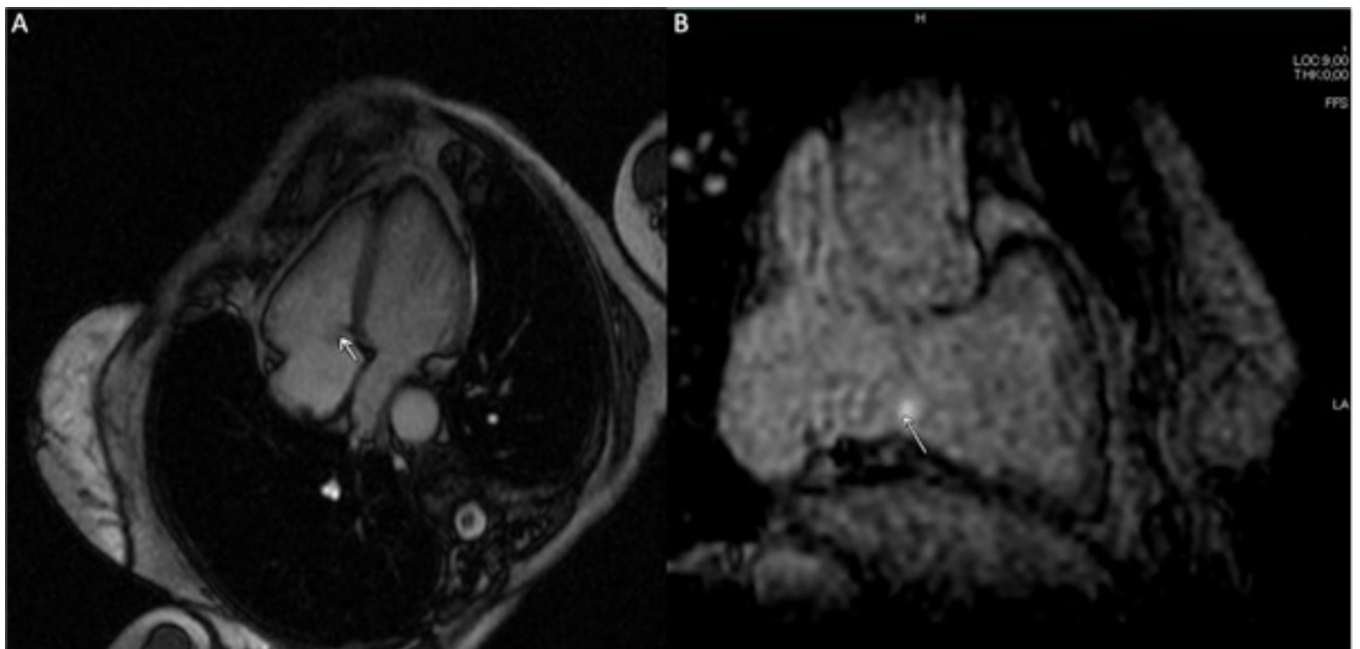


Figure 2 - Cardiac magnetic resonance imaging - Small mass attached to the base of the auricular surface of the septal leaflet of the tricuspid valve (A – A4C view), with tissue characteristics suggestive of papillary fibroelastoma (B – Late gadolinium enhancement).

CC 53

From echocardiographic finding of inferior vena cava mass to renal carcinoma diagnosis and staging

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INTRODUCTION

Inferior vena cava thrombosis (IVCT) is a rare but serious condition. Although asymptomatic in most patients, it can cause leg heaviness, cramping, pain and swelling or abdominal, flank or back pain. Etiologies range from congenital abnormalities to non-congenital causes, the most common being lower extremity deep venous thrombosis and compression of IVC by adjacent structures (neoplasia, abscess, abdominal aortic aneurism, retroperitoneal masses, etc).

CASE PRESENTATION

An 89-years-old woman presented in the emergency department with 1-week symptoms of decompensated heart failure. Her medical history was relevant for arterial hypertension, type 2 diabetes mellitus, dyslipidemia and atrial fibrillation. She was pale and hypertensive at hospital admission and her physical examination was remarkable for a high-pitched, crescendo-decrescendo, midsystolic ejection murmur, best heard at the right upper sternal border radiating to the carotid arteries, decreased breath sounds in the lung bases with crackles in the lower 2/3 of the lung fields and lower limb pitting edema. Decompensated heart failure (HF) due to severe aortic stenosis was suspected and a transthoracic echocardiogram was conducted. SAS was confirmed and a large mass filling the inferior vena cava (IVC) was shown

(Figure 1). A thoracoabdominopelvic computed tomography (TAP CT) was performed and showed a 12x7cm solid expansive lesion of the right kidney with right renal vein and IVC invasion and multiple pathological adenopathies around (Figure 2). Stage III renal carcinoma was assumed. The clinical case was discussed with Vascular Surgery, Urology and Oncology. Attending patient's age, fragility and comorbidities, we chose to maintain hypo-coagulation with DOAC instead of switching to heparin. A renal biopsy for definitive diagnosis was requested. Renal cell carcinoma was confirmed and the patient was considered ineligible for aortic valve replacement.

DISCUSSION

IVCT due to renal cell carcinoma (RCC) occurs only in 9% of cases and implies poor prognosis. Guidelines directing diagnosis and treatment approaches of IVCT are missing but initial management with anticoagulation is recommended for all patients. Additional treatment options, as surgery in RCC, are reserved for selected cases. This case not only highlights the importance of clinical history and multimodality imaging in diagnosis and treatment guidance of IVCT, but also recalls the significance of interprofessional team members discussion and coordination to timely treatment and improve outcomes of this pathology.

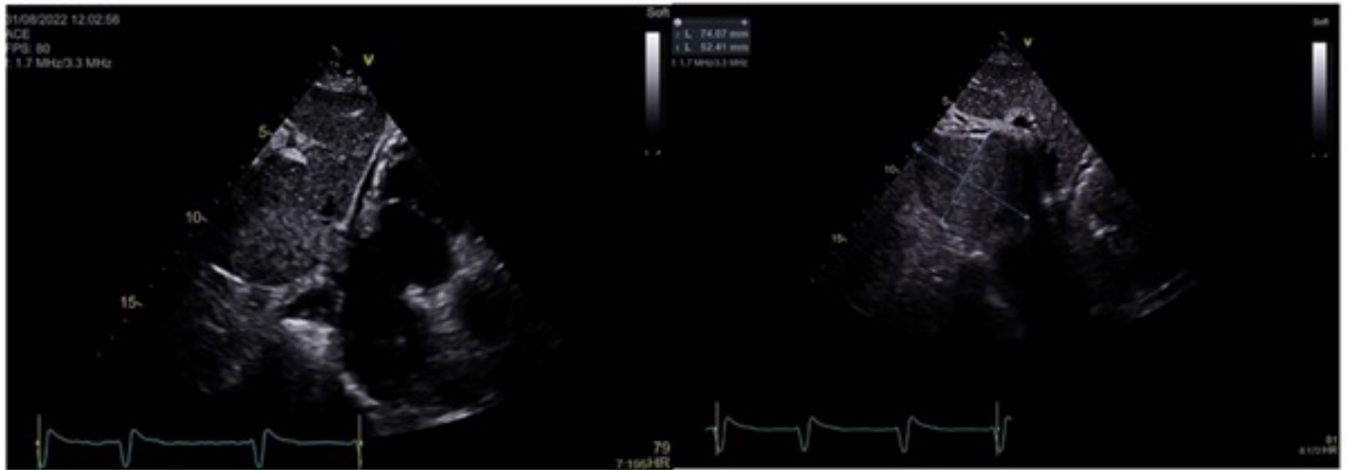


Figure 1 – Transthoracic echocardiogram. Left – Subcostal long-axis view: inferior vena cava mass. Right - Inferior vena cava mass dimensions (74 x 52 mm)

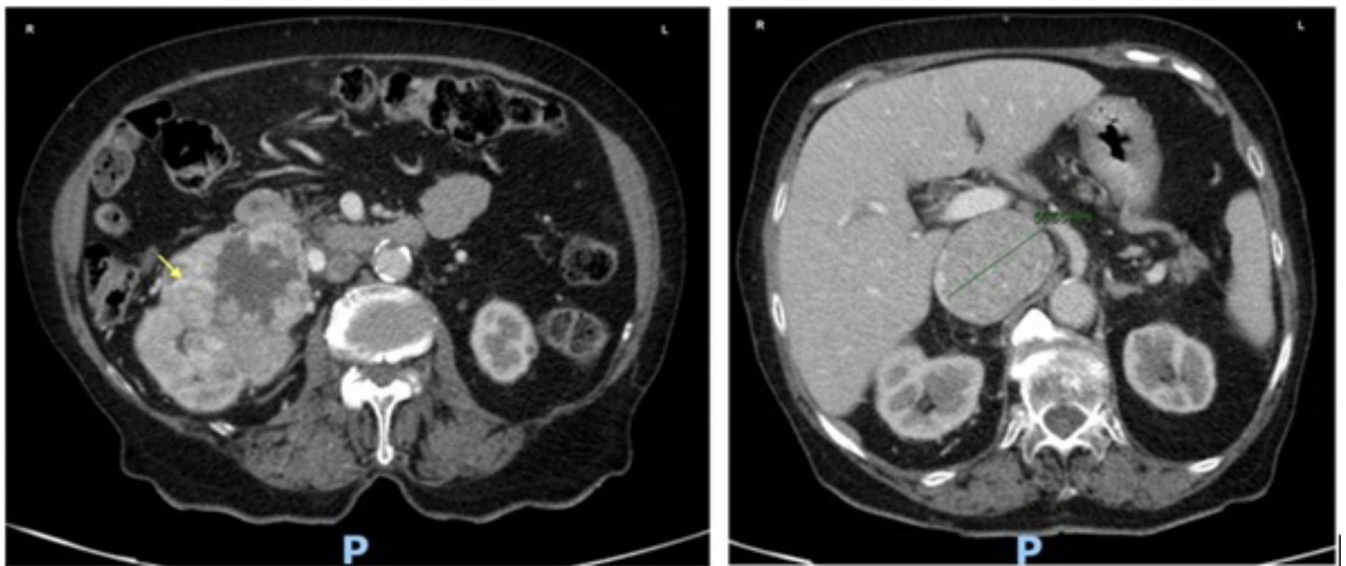


Figure 2 – Abdominal computed-tomography. Left – Renal carcinoma on abdominal computed-tomography. Right – Transversal diameter of IVC mass (55.5mm).

CC 54

The Importance of Multimodality Imaging in the Assessment and Management of Symptoms in Hypertrophic Cardiomyopathy - A Case Report

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INTRODUCTION

Since Hypertrophic Cardiomyopathy is characterized by a wide spectrum of phenotypic expression and clinical manifestations, individualized approaches in the follow-up and management of symptoms and different assessment techniques may be employed. Echocardiography (TTE) remains the first-line technique in the evaluation of HCM patients, but other functional and anatomical tests are crucial in the assessment of their symptoms.

CASE REPORT

A 52-year-old man with HCM presented to the emergency department with angina de novo and dyspnea on exertion. Cardiac auscultation revealed a systolic murmur; there was oedema of the lower limbs.

His medical history included dyslipidemia and HCM with left ventricular outflow tract (LVOT) obstruction in the past, for which he underwent surgical septal myectomy 13 years earlier. He was usually described in NYHA functional class II. A recent TTE showed nonobstructive HCM and preserved biventricular systolic function. The ambulatory ECG showed sinus rhythm with left bundle branch block. He was under bisoprolol 10 mg.

Laboratory results revealed high BNP level. He was admitted to Cardiology department for clarification and management of heart failure and angina symptoms. Low-dose diuretic treatment with furosemide was initiated and, since symptom status and etiology remained unclear, an integrated sequential functional and structural assessment was undertaken.

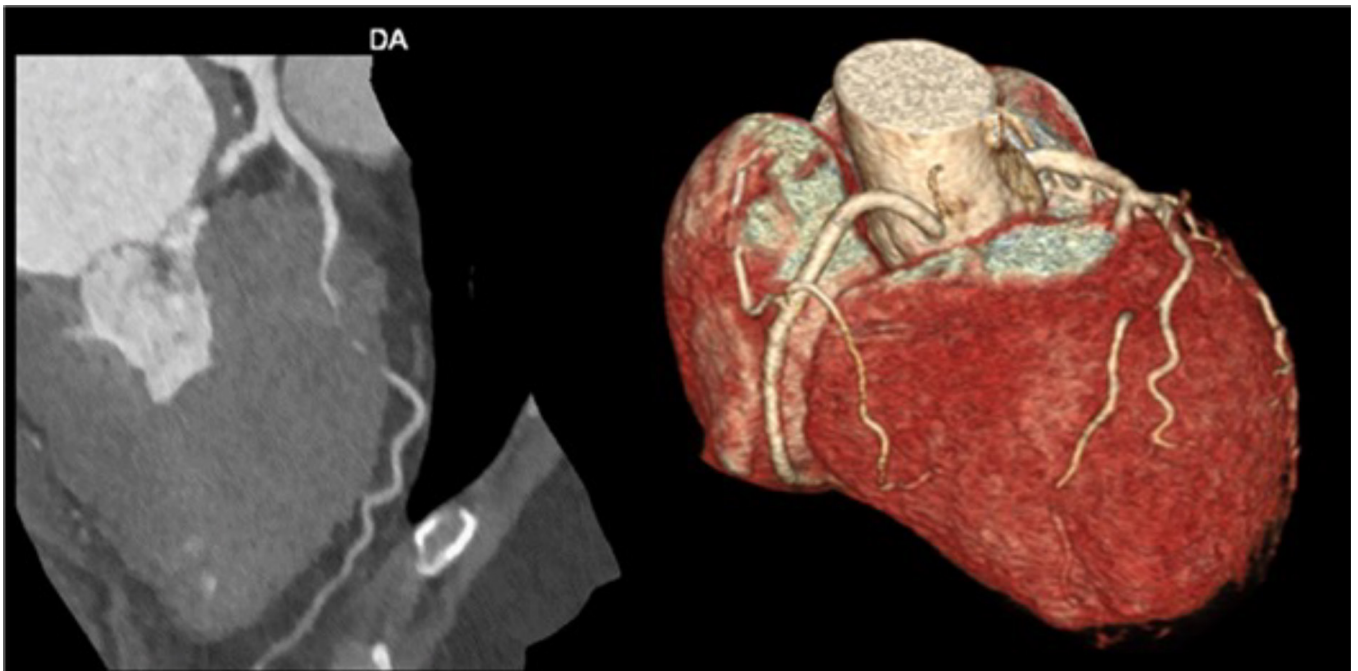
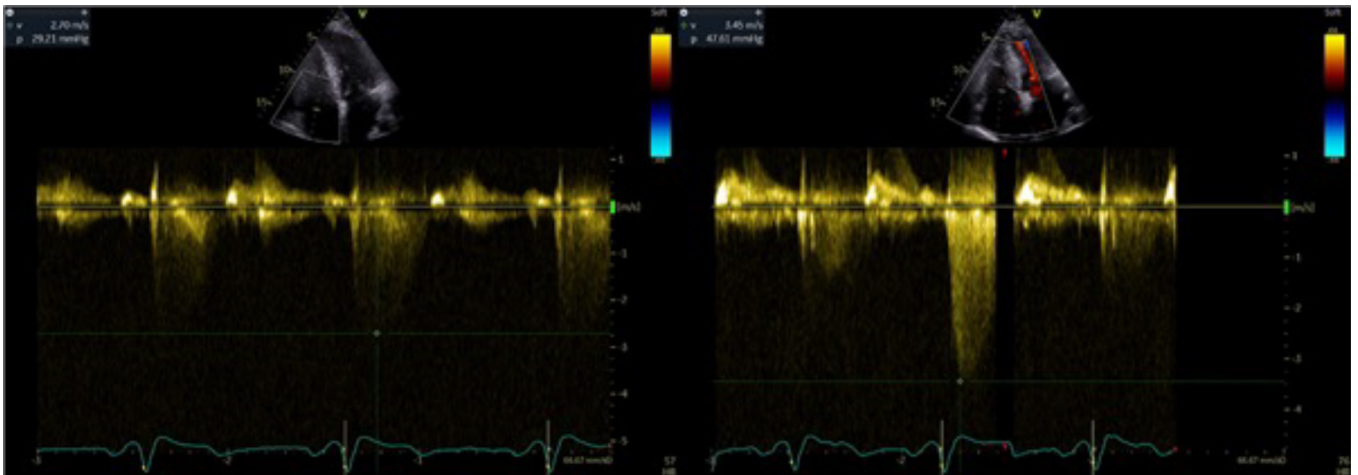
Computed tomography angiography, revealing a left anterior descending artery (LAD) surrounding the apex, with an extensive myocardial bridge encasing the mid segment of the vessel, with almost complete lumen obliteration during systole.

Bicycle ergometry exercise stress echocardiogram demonstrated a paradoxical reduction in the midventricular gradient during exercise, mild SAM aggravated at peak exertion but without resulting in significant LVOT obstruction and worsening of tricuspid regurgitation with exercise (eSPAP 34 mmHg at rest and 53 mmHg at peak exertion).

The worsening pulmonary hypertension with exercise combined with the significant myocardial bridge over LAD seemed likely concurrent causes for the symptoms. Since the patient was already on the maximum tolerated dose of beta-blocker and symptoms persisted, we switched to verapamil with significant clinical improvement and resolution of the angina. In the follow-up consultations after discharge, he remained asymptomatic and without limitation in ordinary daily activity.

DISCUSSION

In the differential diagnosis of heart failure symptoms and chest pain in HCM patients, multimodal approach (in which sequential imaging techniques may be employed depending on individual characteristics and clinical suspicion) has an important role since it can provide answers to specific clinical questions unsolved by individual techniques and help clinicians establish a cause for the symptoms and guide management.



CC 57

Use of fenofibrate in diabetic patients and its impact on renal function- A Clinical Case Report

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In patients with chronic kidney disease (CKD), there is a distinct lipid profile that is characterized by higher levels of triglyceride. This particular lipid profile is associated with subclinical atherosclerosis, coronary artery disease and an increased risk of mortality. Diabetic patients already have an increased risk of CKD due to microvascular complications associated with diabetes. Therefore, it is important to carefully consider the patient's kidney status before prescribing fenofibrate or any other medication. Although generally safe and well tolerated, fibrates initially cause an iatrogenic increase in plasma creatinine and urea. Recent studies show that these changes do not affect patient's renal function and are reversible after stopping fibrate treatment.

JSS, male, 66 years old, retired (electrician), married, 6th year of schooling. Personal history of diabetes mellitus known for 10 years, on oral antidiabetics, diabetic retinopathy, arterial hypertension for 6 years, dyslipidemia, glaucoma, overweight, Ménière's Syndrome. Usually medicated with Metformin + Sitagliptin 1000+50mg, lansoprazole 15mg, telmisartan + hydrochlorothiazide 80+12.5mg, simvastatin + fenofibrate 20+145mg (starting 11/10/2021), beta-histine 24mg, gliclazide 60mg, timolol+dorzolamide 5mg/ml + 20ml. No drug allergies, no smoking habits, sporadic alcohol consumption, sedentary lifestyle. Father died at 60 years with acute myocardial infarction and brother died with ischemic stroke at

61 years. In diabetes surveillance consultation, appears with plasma creatinine values progressively increased since 2018, worsening until 2022. Renal ultrasound showed a small cyst of 17mm, prostate ultrasound, urine and further complementary studies without any changes. On 03/22/2022, metformin + sitagliptin was suspended and linagliptin was prescribed. On 07/29, due to worsening of HbA1c, empagliflozin 10mg was started. Because he maintained azotemia and progressive worsening of HbA1c, he was referred to Nephrology consult. On 09/13/2022, the nephrologist stopped fenofibrate and started atorvastatin 40mg. On 07/02/2023, he was reassessed in consult, with significant improvement in azotemia after discontinuing fenofibrate (pCreatinine 2.6 > 1.6 mg/dL). Since HbA1c remained at 7.6%, it was decided to start metformin 500mg. The patient continues being followed in Nephrology consult and follow-up by the Family Physician.

It is essential to carefully evaluate the risk-benefit ratio of drug prescription in diabetic patients. Regular monitoring of renal function and adoption of preventive measures should be implemented to minimize the risks associated with the use of drugs such as fenofibrate. The clinical case highlights the importance of correct and effective medication management in patients with multiple comorbidities, always paying attention to possible drug iatrogenesis.

13TH CHALLENGES IN CARDIOLOGY

POSTERS



PO 03

Recurrence of acute coronary syndrome in the elderly: predictors and prognosis - insights from a multicentre national registry

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The burden of cardiovascular (CV) disease remains at the top of society's health problems, with acute coronary syndromes (ACS) undoubtedly being one of the major causes. Patients with a known history of CVD are at increased risk of recurrent events. However, this recurrence's incidence rate and clinical impact vary between populations. Elderly patients represent a large proportion of patients admitted for a second ACS. The decision on its management lacks support in clinical guidelines, as studies generally exclude patients with significant comorbidities and tend to underrepresent this group.

This study aimed to analyse the characteristics and outcomes of elderly patients admitted with ACS with a previous history of myocardial infarction (MI) versus those who did not.

The authors performed a retrospective analysis of patients older than 75 years included in the Portuguese Registry on Acute Coronary Syndromes (ProACS) between 2010 and 2022. Comparison tests were performed. A COX proportional hazard regression method was applied to generate the proposed risk models. A p-value less than 0.05 was considered statistically significant.

A total of 8658 patients older than 75 years admitted for ACS were analysed, 6630 (76.6%) without a history of MI (group 1) and 2028 (23.4%) with a previous MI. Table 1 shows the most important baseline characteristics and differences between groups. Table 2 demonstrates the group differences regarding admission diagnosis, complementary study results and treatment. Table 3 represents in-hospital complications. At discharge, 16.5% of group 1 and 21.2% of group 2 had a planned cardiac rehabilitation program (CRP) (p-value=0.01). At 1-year follow-up, 15.6% of patients with more than 75 years admitted for ACS died and 17.4% were re-hospitalized by a CV cause. COX-regression analysis identified the history of MI as a predictor factor of CV re-hospitalization (HR 1.43; 95%CI [1.10;1.86], p-value<0.01) and of the composite of mortality and CV re-hospitalization (HR 1.37; 95%CI [1.10;1.71], p-value<0.01).

In conclusion, the elderly with a second episode of ACS tends to have more CV risk factors and comorbidities than those without a MI history. Furthermore, the recurrence of MI seems to be an independent predictor of 1-year re-hospitalization and the composite of mortality and CV re-hospitalization. Then, these results should increase the awareness of this group of patients.

TABLE 1. BASELINE CHARACTERISTICS

	Overall (N=8658)	Group 1 (N=6630)	Group 2 (N=2028)	p-value
Male, n (%)	5001/8658 (57.8%)	3645/6630 (55.0%)	1356/2028 (66.9%)	<0.01
Age in years, mean (dp)	81.4 (4.8)	81.5 (4.8)	81.2 (4.7)	0.01
Body mass index in kg/m ² , mean (dp)	26.7 (4.1)	26.6 (4.1)	26.8 (4.1)	0.04
Smoker, n (%)	358/8650 (4.1%)	279/6622 (4.2%)	79/2028 (3.9)	0.53
Hypertension, n (%)	7235/8564 (84.5%)	5416/6549 (82.7%)	1819/2015 (90.3%)	<0.01
Dyslipidemia, n (%)	4966/8249 (60.2%)	3446/6267 (55.0%)	1520/1982 (76.7%)	<0.01
Diabetes, n (%)	3221/8543 (37.7%)	2263/6530 (34.7%)	958/2013 (47.6%)	<0.01
Valvular disease, n (%)	614/8479 (7.2%)	408/6494 (6.3%)	206/1985 (10.4%)	<0.01
Heart failure, n (%)	958/8518 (11.2%)	533/6529 (8.2%)	425/1989 (21.4%)	<0.01
Chronic Kidney disease, n (%)	1033/8056 (12.8%)	650/6185 (10.5%)	383/1871 (20.5%)	<0.01

TABLE 2. DIAGNOSIS, COMPLEMENTARY TESTS RESULTS AND TREATMENT

	Overall (N=8658)	Group 1 (N=6630)	Group 2 (N=2028)	p-value
ADMISSION DIAGNOSIS				
STEMI, n(%)	3180/8658 (36.7%)	2794/6630 (42.1%)	386/2028 (19.0%)	<0.01
NSTEMI, n (%)	4436/8658 (51.2%)	3161/6630 (47.7%)	1275/2028 (62.9%)	<0.01
Angina instável, n (%)	499/8658 (5.8%)	316/6630 (4.8%)	183/2028 (9.0%)	<0.01
Undefined MI, n (%)	543/8658 (6.3%)	359/6630 (5.4%)	184/2028 (9.1%)	<0.01
LABORATORY RESULTS				
Hemoglobin at admission in g/dL, mean (dp)	12.8 (1.9)	12.8 (1.8)	12.6 (1.9)	<0.01
Serum creatinine at admission in mg/dL ¹ , median (P25, P75)	1.1 (0.9;1.4)	1.0 (0.8;1.4)	1.2 (0.9;1.6)	<0.01
Maximum serum creatinine in mg/dL ¹ , median (P25, P75)	1.3 (1.0;1.9)	1.3 (1.0;1.8)	1.4 (1.1;2.0)	<0.01
HbA1c in % ¹ , median (P25, P75)	6.1 (5.6;7.0)	6.0 (5.6;6.8)	6.2 (5.7;7.3)	<0.01
HbA1c >6.5%, n (%)	963/2728 (35.3%)	715/2127 (33.6%)	428/601 (41.3%)	<0.01
LDL cholesterol in mg/dL, mean (dp)	101 (37)	104 (36)	92 (37)	<0.01
ANGIOGRAPHIC PROFILE AND REVASCULARIZATION TREATMENT				
Left heart catheterization performed, n (%)	6323/8593 (73.6%)	4986/6577 (75.8%)	1337/2016 (66.3%)	<0.01
Culprit coronary artery				
Left main, n (%)	120/4525 (2.7%)	95/3542 (2.7%)	25/983 (2.5%)	0.81
Anterior descending artery, n (%)	1645/4525 (36.4%)	1370/3542 (38.7%)	275/983 (28.0%)	<0.01
Circumflex artery, n (%)	646/4525 (14.3%)	501/3542 (14.1%)	145/983 (14.8%)	0.63
Right coronary artery, n (%)	1111/4525 (24.6%)	908/3542 (25.6%)	203/983 (20.7%)	<0.01
Coronary artery bypass, n (%)	102/4525 (2.3%)	31/3542 (0.9%)	71/983 (7.2%)	<0.01
Multivessel disease ² , n (%)	3221/5580 (57.7%)	2431/4426 (54.9%)	790/1154 (68.5%)	<0.01
PCI, n (%)	4545/8593 (52.9%)	1685/2789 (60.4%)	222/384 (57.8%)	<0.01
CABG ³ , n (%)	442/8582 (5.2%)	353/6569 (5.4%)	89/2013 (4.4%)	0.09
LEFT VENTRICULAR SYSTOLIC FUNCTION				
LVEF in %, mean (dp)	49 (13)	50 (13)	47 (12)	<0.01

STEMI - ST-elevation myocardial infarction; NSTEMI - non-ST-elevation myocardial infarction; MI - myocardial infarction; HbA1c - glycated hemoglobin; LDL - low-density lipoprotein; PCI - percutaneous coronary intervention; CABG - coronary artery bypass graft; GP - glycoprotein; LVEF - left ventricular ejection fraction

TABLE 3. IN-HOSPITAL COMPLICATIONS

	Overall (N=8658)	Group 1 (N=6630)	Group 2 (N=2028)	p-value
Acute heart failure, n(%)	2107/8221 (25.6%)	1566/6274 (25.0%)	541/1947 (27.8%)	0.01
Cardiogenic shock, n (%)	514/2180 (23.6%)	412/1620 (25.4%)	102/560 (18.2%)	<0.01
Mechanical complication, n (%)	87/8223 (1.1%)	83/6276 (1.3%)	4/1947 (0.2%)	<0.01
Stroke, n (%)	78/8220 (0.9%)	66/6273 (1.1%)	12/1947 (0.6%)	0.08
Major haemorrhage, n (%)	89/8109 (1.1%)	64/6188 (1.0%)	25/1921 (1.3%)	0.33
In-hospital mortality, n (%)	643/8490 (7.6%)	511/6492 (7.9%)	132/1998 (6.6%)	0.06

PO 04

Impact of phase II of a cardiac rehabilitation program in older adults with coronary artery disease - a retrospective observational study

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Cardiac rehabilitation (CR) is an evidence-based secondary prevention intervention aimed at optimising cardiovascular (CV) health. The main goals of CR are to improve modifiable CV risk factors, increase functional capacity, and reduce morbidity and mortality. Nonetheless, the underuse of CR in all ages remains a major concern, particularly described among older adults.

This study aimed to investigate the effects of phase II hybrid cardiac rehabilitation program (CRP) on physical capacity and control of CV risk factors among coronary artery disease patients and compare their benefits between younger and older patients.

A retrospective study included patients who had completed the phase II CRP between 2017 and 2022. Patient selection and information collection were obtained through medical records. Outcomes were International Physical Activity Questionnaire (IPAQ) results, estimated functional capacity in the cardiac stress test, lipid profile, glycated hemoglobin (HbA1c) and body mass index. Patients were divided into two groups: group 1 for patients aged 18 to 59 years and group 2 for those older than 60. Variables were analysed at the beginning (T0) and the end (T1) of phase

II. Group comparison tests were performed. A p-value less than 0.05 was statistically significant. Statistical analysis was performed using SPSS software v25.0.

One hundred and fifty-eight patients were enrolled in phase II CRP, and 126 completed it, accounting for adherence of 79.2% (n=76) in group 1 and 86.2% (n=50) in group 2 (p-value=0.27). Regarding baseline characteristics (Table 1), we observed a significant difference in arterial hypertension (p-value=0.01) and diabetes (p-value=0.01), more prevalent in older patients, and smoking in younger ones (p-value=0.03). Except for the IPAQ results in group 2 and HbA1c in both groups, all other variables had a significant difference between T0 and T1 in both groups. However, there is no significant difference when comparing the results between groups (Table 2).

In conclusion, the benefits of phase II CRP on physical capacity and control of CV risk factors are equivalent in both age groups. Despite this, there is an underrepresentation of patients aged over 60 years, in line with other observational analyses, which should alert clinicians to a greater referral of this group.

TABLE 1. BASELINE CHARACTERISTICS

	Overall (n=126)	Group 1 (n=76)	Group 2 (n=50)	p-value
DEMOGRAPHIC CHARACTERISTICS				
Male, n (%)	108 (85.7)	67 (88.2)	41 (82.0)	0.33
Arterial hypertension, n (%)	79 (62.7)	37 (48.7)	42 (84.0)	<0.01
Diabetes, n (%)	26 (20.6)	9 (11.8)	17 (34.0)	<0.01
Dyslipidemia, n (%)	102 (81.0)	62 (81.6)	40 (80.0)	0.83
OSAHS, n (%)	30 (20.6)	15 (19.7)	15 (30.0)	0.19
Current smoker, n (%)	63 (50)	44 (57.9)	19 (38.0)	0.03
Former smoker, n (%)	33 (26.2)	17 (22.4)	16 (32.0)	0.23
ADMISSION DIAGNOSIS				
STEMI, n (%)	50 (39.7)	27 (35.5)	23 (46.0)	0.24
NSTEMI, n (%)	50 (39.7)	27 (35.5)	23 (46.0)	0.24
Unstable angina, n (%)	1 (0.8)	1 (1.3)	0 (0.0)	1.00
Other, n (%)	2 (1.6)	0 (0.0)	2 (4.0)	0.16

OSAHS - Obstructive sleep apnea/hypopnea syndrome; STEMI - ST-elevation myocardial infarction; NSTEMI - non-ST-elevation myocardial infarction

TABLE 2. RESULTS AND GROUP COMPARISON

	Group 1 (n=44)				Group 2 (n=25)				p-value ($\Delta 1/\Delta 2$)
	T0	T1	p-value (T0/T1)	$\Delta 1$ T1-T0	T0	T1	p-value (T0/T1)	$\Delta 2$ T1-T0	
CLINICAL EVALUATION									
BMI in kg/m ² , mean (SD)	28.4 (4.3)	24.5 (5.2)	<0.01	(-) 3.6 (3.3)	29.9 (14.2)	22.7 (3.6)	<0.01	(-) 6.8 (12.9)	0.06
Smoking cessation, n (%) ¹	NA	26 (70.3)	NA	NA	NA	8 (47.1)	NA	NA	0.10
EXERCISE CAPACITY									
IPAQ in METs, median (IQR)	792.0 (1247.0)	1386.0 (2247.0)	<0.01	(+) 614.0 (2243.5)	735.0 (1408.0)	990.0 (1634.0)	0.25	(+) 469.0 (1325.3)	0.14
Exercise testing capacity in METs, mean (SD)	12.0 (3.2)	14.0 (3.1)	<0.01	(+) 1.4 (3.2)	10.2 (3.2)	11.8 (3.9)	0.01	(+) 1.1 (2.8)	0.66
LAB TESTS RESULTS									
LDL-c in mg/dL, mean (SD)	125.7 (41.2)	61.4 (25.3)	<0.01	(-) 58.4 (42.1)	117.4 (49.8)	60.4 (22.5)	<0.01	(-) 44.3 (49.7)	0.11
HDL-c in mg/dL, mean (SD)	44.1 (11.5)	45.8 (12.2)	<0.01	(+) 2.8 (7.7)	45.5 (11.4)	47.7 (10.8)	0.14	(+) 2.0 (8.8)	0.62
TG in mg/dL, mean (SD)	162.4 (70.7)	106.1 (55.4)	<0.01	(-) 52.2 (67.9)	157.4 (72.8)	106.0 (36.2)	<0.01	(-) 46.7 (68.1)	0.68
HbA1C in %, mean (SD)	5.7 (0.7)	5.6 (0.7)	0.92	(-) 0.01 (0.6)	6.2 (1.7)	5.8 (0.6)	0.14	(-) 0.3 (1.2)	0.18

BMI - body mass index; IPAQ - International Physical Activity Questionnaire; METs - metabolic equivalents; LDL-c - low-density lipoprotein cholesterol; HDL-c - high-density lipoprotein cholesterol; HbA1C - glycated hemoglobin

¹ Of the total of smoker patients (n=37, 7 missing [group1]; n=17, 2 missing [group2]).

PO 12

ASSIMETRIC LEFT VENTRICULAR HYPERTROPHY IN PATIENTS WITH SEVERE AORTIC STENOSIS: PREVALENCE AND SIGNIFICANCE

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BACKGROUND

Cardiac magnetic resonance (CMR) is the gold standard non-invasive method for the quantification of left ventricular (LV) mass and volume. In patients with severe aortic stenosis (AS), this imaging method provides the characterization of LV remodeling. Distinct patterns of LV adaptation, beyond classical concentric LV hypertrophy, have been described.

AIM

To assess the prevalence and significance of asymmetric LV hypertrophy in patients with severe AS referred for surgical aortic valve replacement.

METHODS

Single center prospective study of patients with severe symptomatic AS referred for surgical AVR, with no previous history of cardiomyopathy. Before surgery, all patients underwent a transthoracic echocardiography and CMR for LV assessment and tissue characterization. As described, LV remodeling was characterized according to LV mass, volumes and function, both before and the 3rd to 6th month after AVR by CMR - figure 1A. Asymmetric LV wall thickening was defined as a regional wall thickening ≥ 13 mm (from the short-axis views of the LV in end diastole, with exclusion of LV trabeculations) that was also >1.5 -fold the thickness of the opposing myocardial segments, on at-least two adjacent short-axis slices. Clinical and imaging data were compared in patients with and without asymmetric wall thickening.

RESULTS

130 consecutive patients (72years [70-73years], 48% men] with severe AS were included: mean transaortic pressure gradient (AVmean): 61 ± 18 mmHg; aortic valve area (AVA): 0.7 ± 0.2 cm²; mean LV ejection fraction (CMR): $60 \pm 10\%$; mean LV indexed stroke volume: 47 ± 11 ml; mean LV indexed mass [LVmi]: 82 ± 27 g/m². Concentric remodeling and hypertrophy were the most frequent LV remodeling patterns (25% and 49% of the patients, respectively). Asymmetric LV thickening was present in 71 patients (55% of the cohort), being predominant at mid inferior and basal anterior septum - Figure 1B. None of the patients had significant pre-operative intra-ventricular gradient. Patients with asymmetric wall thickening had significantly higher BNP levels ($p=0,043$), higher valvular gradients ($p=0,028$) and by CMR: worst LV function ($p=0,03$), higher LVmi ($p<0,001$) and LGE mass ($p=0,036$) - figure 1C.

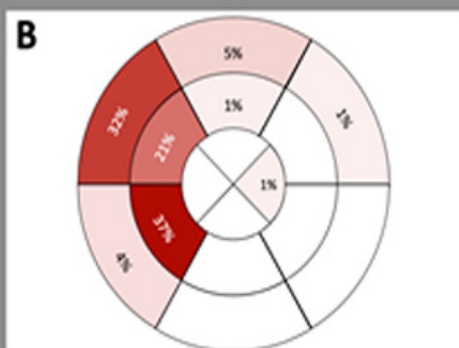
CONCLUSIONS

In this cohort of patients with classical severe aortic stenosis, asymmetric wall thickening was frequent, with predominant basal e mid septum involvement, being associated with worse indexes of both valve narrowing and LV function and remodeling.

A

	LVM	LVEDV	M/V	EF
Normal LV	=	=	=	=
Concentric remodeling	=	↓	↑	+/↑
Concentric LVH	↑	=	↑	+/↓
Eccentric LVH	↑	↑	=	=
Adverse remodeling	↑	↑	=	↓

	LVM (g/m ²)	LVEDV (ml/m ²)	M/V
Male	≤ 85	≤ 108	≤ 0,9
Female	≤ 68	≤ 96	≤ 0,8



C

	Asymmetric pattern n=71	No-asymmetric pattern n= 59	p-value
Baseline characteristics			
Troponin (ng/L)	15 [11 - 22]	10 [7 - 14]	p<0,001
NTproBNP (ng/mL)	675 [148 - 1935]	525 [175 - 1210]	p=0,043
TTE characteristics			
AV mean (mmHg)	64 ± 18,4	57,4 ± 16,1	p=0,028
Global longitudinal strain (%)	- 13,8 ± 3,6	- 15,8 ± 3,8	p=0,005
CMR characteristics			
LVEF, CMR (%)	59 [52 - 65]	63 [56 - 69]	p=0,030
LV indexed mass (g/m ²)	90,6 ± 27,8	71,2 ± 21,1	p<0,001
Max wall thickness (mm)	15,9 ± 2,2	12,2 ± 2,1	p<0,001
Indexed LVEDV (mL/m ²)	89,5 ± 24,6	81,2 ± 20,3	p=0,042
LGE mass (g)	4 [0,7 - 10,3]	1,9 [0,1 - 5,9]	p=0,036

Figure 1 – A: CMR patterns of LV hypertrophy; **B:** Site of maximal wall thickening in asymmetric pattern based on the 17-segment model of the LV; **C:** Comparative analyses of patient characteristics between those with and without asymmetric pattern in aortic stenosis.

PO 13

Time is of the essence: the importance of repeated echocardiography before discharge in anterior wall acute myocardial infarction

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INTRODUCTION

The incidence of left ventricular (LV) thrombus as a complication of anterior wall acute myocardial infarction (AMI) has declined substantially since the introduction of re-perfusion therapies, but it still remains an important complication. Only a minority can be detected within the first 24 hours, with most thrombi developing between 72 hours and 2 weeks after AMI. Thus, timing of imaging appears to have diagnostic importance. Larger infarcts, anteroapical wall motion abnormalities, and reduced ejection fraction are considered to be risk factors. In these cases, repeat echocardiography should be considered if no thrombus is detected on early imaging.

GOALS

Determining if repeat echocardiography prior to discharge in anterior wall AMI increases the detection rate of LV thrombus.

METHODS

Retrospective, observational and analytical study, including two groups of patients admitted for anterior wall AMI: group 1: admitted between 1st of January and 31st of December 2015 and group 2: admitted between 1st of November 2021 and 30th of November 2022. The reason for choosing these two time intervals was due to the informal implementation of a different screening strategy for LV thrombus in patients admitted for anterior wall MI. In more recent years, particularly since late 2021, an attempt has been made to repeat echocardiography prior to discharge in patients considered to be at higher risk, although no formal screening protocol has been implemented yet.

Data regarding patients characteristics, laboratory findings and echocardiographic parameters was collected. To ensure an even distribution of confounders between groups and therefore increase between group comparability, propensity score matching was performed.

RESULTS

Initially, 199 patients were selected. Propensity score matching was performed in order to account for differences in baseline characteristics between groups. A total of 44 patients from each group were included in the final analyses. In group 1, 8 patients had repeated echocardiography prior to discharge and 4 patients were diagnosed with LV thrombus, 2 of which were detected on the second exam. In group 2, 34 patients had repeated echocardiography prior to discharge and 8 were diagnosed with LV thrombus, all detected on the second echocardiogram. There were no statistically significant differences in the number of LV thrombi diagnosed in the two groups (χ^2 (1, $N=88$) = 0,192 , p = 0,661).

CONCLUSION

The lack of statistically significant differences in the number of LV thrombi between the two groups may be due to an incomplete selection of high-risk patients, due to the lack of well-defined selection criteria. More importantly, in group 2, if it had not been for repeat echocardiogram prior to discharge, no LV thrombi would have been detected. We believe this further supports the need for an established screening protocol systematically targeting patients at higher risk.

PO 17

Diagnostic role of high-sensitivity cardiac troponin: is it a good rule-out test for unstable angina? - a single centre analysis

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BACKGROUND

Unstable angina (UA) is defined as myocardial ischemia at minimal exertion or at rest without myocardial injury. According to current guidelines, fewer UA have been diagnosed after introduction of high-sensitivity (hs) cardiac troponin (cTn) assays. Furthermore, these patients have a low risk of cardiovascular events and the optimal approach regarding selecting patients that benefit from non-elective invasive testing is not well established.

The purpose of this analysis was to determine the usefulness of hs cTn in discriminating coronary artery disease (CAD) in UA.

MATERIAL AND METHODS

Retrospective single-centre subanalysis of 166 UA patients admitted for invasive stratification from 2015 to 2022. Two cohorts were defined according to the cTn assay used (hs-TnI vs conventional TnI) and its baseline characteristics, coronary angiography findings and associated extended major adverse cardiovascular events (MACE) compared. Receiver-operating characteristic (ROC) analysis was used to determine diagnostic accuracy of several variables.

RESULTS

Overall, mean age was 64 ± 11 years, 72% were male, significant CAD was diagnosed in 50%, the incidence of MACE was 14% and no differences between groups were found, except for dyslipidemia ($p=0.029$). Seventy-two UA cases (43%) were diagnosed using hs-cTn assay. After ROC analysis of this group, the diagnostic accuracy of hs-cTn for the presence of significant CAD was higher for maximum levels of hs-cTn - with adequate discriminatory power (area under the curve [AUC] 0.778; 95% CI: 0.671-0.884) - although being lower for initial values or absolute change in troponin levels (AUC 0.626 and 0.751, respectively). The cutoff value of 13.35 ng/dL for maximum levels of hs-cTn predicted CAD with a sensitivity of 80% and a specificity of 62%. Other variables, such as NT-proBNP or creatinine had low discriminatory power (AUC 0.552 and 0.582, respectively).

CONCLUSIONS

In our population of UA patients, clinical characteristics, CAD and MACE did not differ significantly regardless of the use of hs or non hs-cTn assays. Although maximum hs-cTn had an adequate discriminatory power for CAD, this was not the case for other variables. Therefore, other variables should be considered in addition to initial hs-cTn to better select those that may benefit from non-elective invasive stratification.

PO 18

Sex-based differences in ST-segment elevation myocardial infarction: a multicentre national registry analysis

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BACKGROUND

Sex differences in ST-segment elevation myocardial infarction (STEMI) are not fully understood. Female patients appear to have higher mortality.

PURPOSE

To establish sex-differences in Portuguese STEMI patients.

MATERIAL AND METHODS

Retrospective multicentre analysis of STEMI patients included in the Portuguese Registry on Acute Coronary Syndromes (ProACS) between October 2010 and October 2022. Two cohorts were defined according to sex. Baseline characteristics, clinical findings, treatment and mortality were compared. Multivariate analysis was performed to assess predictors of mortality.

RESULTS

A total of 14470 patients were included with a mean age of 64 ± 14 years, of which 26% were female. Female patients were significantly older ($p < 0.001$) and with higher prevalence of several cardiovascular risk factors such as: high blood pressure ($p < 0.001$), diabetes ($p < 0.001$) and dyslipidemia ($p = 0.015$), as well as higher reported past medical history, namely ischemic stroke/transient ischemic attack ($p = 0.001$), renal disease ($p < 0.001$) and dementia ($p < 0.001$). On the other hand,

previous coronary artery disease was less common comparing to men. Reperfusion therapy was less frequent in females ($p < 0.001$), with less cases of multivessel disease ($p = 0.004$). Regarding inpatient medical treatment women were less frequently prescribed medical therapy and more frequently needed inotropes ($p < 0.001$). Regarding discharge medication, similar tendencies were observed, and women were less frequently referred to cardiac rehabilitation programs ($p < 0.001$).

Concerning prognosis, women had more complications while at hospital, namely, congestive heart failure ($p < 0.001$), ischemic stroke ($p < 0.001$) and intra-hospital mortality ($p < 0.001$). Similarly, women had higher thirty-day and one-year mortality ($p < 0.001$) and non-cardiac hospital readmission ($p < 0.001$). After multivariate analysis, female sex (OR=1.633; CI 95% [1.065-2.504]; $p = 0.025$) remained as an independent factor for intra-hospital mortality but not for thirty-day and one-year mortality.

CONCLUSIONS

In our population, female patients had statistically significant differences in comparison to men regarding clinical characteristics, treatment and prognosis. Nevertheless, female sex was an independent risk factor only for intra-hospital mortality.

PO 20

Heart Failure through patient's voices

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INTRODUCTION

Getting to know patients with Heart Failure in more depth and the impacts that the disease has on their lives will allow creating more effective and targeted follow-up strategies for these patients, in order to improve their quality of life and the way they manage the disease.

OBJECTIVES

Evaluate HF patient attributes, perceptions about their own condition, as well as assess their needs, expectations and satisfaction to NHS Healthcare.

MATERIAL AND METHODS

Market Research study with a national representation, consisting of two different phases. Phase 1: Qualitative component using CATI system (*Computer Assisted Telephone Interviews*), involving 20 HF patients. Phase 2: Quantitative component using an online survey through a CAWI system (*Computer Assisted Web Interview*) with a sample of 117 HF patients.

The patients were recruited by AADIC, Spirituc, Centers activation and media divulgation

CONCLUSIONS

Diagnosis of HF is mostly done in consultations and routine examinations and patients are followed up in more than $\frac{3}{4}$ of cases in the public setting.

Although after diagnosis the level of knowledge about the disease increases slightly, at the time of diagnosis, levels of information about the disease are frankly low.

One third of patients are followed up for HF only every six months. 70% of respondents had an episode of decompensation in the last two years.

The main impacts of the disease are felt in the personal sphere of patients.

Most relevant challenges along the course of the HF are linked to overcoming the fear/anxiety/depression and dealing with physical symptoms, such as pain, shortness of breath and fatigue.

The main frustrations are related to physical restriction, loss of autonomy and psychological impacts, and for last, the expectations with regards to HF are focused on new and more effective therapies.

Based on these data, it is essential to reinforce the follow-up of these patients in the setting of public health institutions to improve the follow-up given to patients and reduce the interval between follow-up moments, which could have a positive impact on reducing the number of situations of decompensation.

Additionally, it is also important to reinforce the psychological follow-up given to these patients, so that they can deal better, not only with the disease, but also with the consequences that it has on the lives of these patients.

Finally, it is important to increase awareness and knowledge about the disease, so that patients are aware of the symptoms, which could increase the number of diagnoses and anticipate them.

PO 23

Early treatment of heart failure with dapagliflozin in Portugal: population baseline characteristics of a non-interventional observational study (EVOLUTION-HF)

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INTRODUCTION

Heart failure (HF) is one of the most important chronic health conditions worldwide, causing considerable healthcare-related costs and a high burden on patients, national health services, and society. In the DAPA-HF study, dapagliflozin decreased the risk of hospitalisation for HF and mortality in patients with HF with reduced ejection fraction (HFrEF). Little is known about treatment profiles and characteristics of patients initiating dapagliflozin for HFrEF in clinical practice. The EVOLUTION-HF study programme aims to assess treatment patterns and patient reported outcomes in patients initiating dapagliflozin for HFrEF in the real world setting in 13 countries.

OBJECTIVES

This preliminary analysis of the EVOLUTION-HF Portugal study aimed to describe baseline demographic and clinical characteristics of patients initiating treatment with dapagliflozin for HFrEF in the retrospective cohort.

MATERIAL AND METHODS

EVOLUTION-HF is an observational, longitudinal cohort study including adult patients with a confirmed diagnosis of HFrEF who are initiated on dapagliflozin (both outpatient

and inpatient settings) between 1st March and 31st October 2021 in 6 cardiology clinics distributed alongside the country. Patients with a diagnosis of Type 1 Diabetes or previous SGLT2i treatment cannot enter the study. Study index is the date of dapagliflozin initiation and patients are followed for a period of 12-months or until study discontinuation/loss to follow-up or death, whichever occurs first.

RESULTS

107 patients were included, 71% male. Mean (SD) age was 67 (12) years and mean BMI (SD) was 27.3 (5.1) Kg/m². Mean (SD) eGFR was 70.4 (22.8) mL/min/1.73 m² and median (IQR) NT-proBNP was 1038 (1662) pg/mL. Main cause of HF was non-ischemic in 49% of the cases. Most patients were on NYHA class II (86%). Mean (SD) LVEF was 30% (6.3). Of the patients that were not in sinus rhythm (26%), 82% had auricular fibrillation, and 9% auricular flutter. Most frequent abnormalities found in the electrocardiogram were non-specific repolarization changes (28%) and complete left bundle branch block (13.5%). In the 12-month period prior to the index date, 19% of the patients were hospitalised for HF. Regarding medical history, 68% had hypertension, 65% dyslipidemia, 35% myocardial infarction, 28% an implantable cardioverter-defibrillator and 27% atrial fibrillation. The majority of the patients were treated with beta blocker (97%), aldosterone antagonist (78.5%) and angiotensin receptor-neprilysin inhibitor (67.3%).

CONCLUSIONS

The characteristics of patients initiating dapagliflozin in clinical practice in Portugal are quite similar to the DAPA-HF population. However, EVOLUTION-HF population presents with a slightly higher proportion of patients in NYHA II and lower median NT-proBNP. The EVOLUTION-HF study will provide novel data on real-world use of dapagliflozin in the HFrEF setting.

PO 30

Revitalizing Right Ventricular Function: Unraveling the Therapeutic Potential of Levosimendan

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BACKGROUND

Due to its high morbidity and mortality, acute heart failure still presents a therapeutic challenge despite recent advancements. Particularly, concomitant failure of the right ventricle is an independent prognostic marker for mortality in patients with acute decompensated heart failure in recent studies. Levosimendan exerts positive inotropic and vasodilatory effects, with the ability to improve left heart function in patients with heart dysfunction. However, its effects on right heart function are still poorly understood.

OBJECTIVE

The aim of this study is to perform a quantitative assessment of right ventricular (RV) function after administration of intravenous (IV) levosimendan in patients hospitalized in the cardiac intensive care unit (CICU) due to acute decompensated heart failure with reduced left ventricular function (HFrEF).

METHODS

We retrospectively analyzed 54 patients admitted to a CICU due to ADHF. Echocardiography measurements were performed during hospitalization and

compared between patients that received 24-h intravenous (IV) levosimendan and patients that did not.

RESULTS

Baseline characteristics were comparable between the two groups. 25 patients (23.8%) received IV levosimendan during CICU hospitalization. We found that infusion of levosimendan was associated with significant improvements of RV function: right ventricular fractional area change (22 ± 2 vs. 18 ± 2 , $p < 0.001$), tricuspid annular plane systolic excursion (10.5 ± 2.5 vs. 8.5 ± 0.19 , $p = 0.02$), RV fractional area change (26 ± 4 vs. 21 ± 2 , $p < 0.001$), were significantly higher, and systolic pulmonary artery estimated pressure (43 ± 5 vs. 54 ± 7 , $p < 0.001$) was lower in the levosimendan group than in the control group.

CONCLUSIONS

In patients hospitalized with acute decompensated HFrEF, the administration of levosimendan during CICU stay is associated with improvement of RV function. Further large-scale studies are needed to verify the positive effects of this drug.

PO 37

Unstable angina in the COVID era - a single-center retrospective cohort study

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INTRODUCTION

The year 2020 was marked by the COVID pandemic and the first case in Portugal was reported on March 2nd. This posed a challenge to healthcare infrastructures, with the primary goal of minimizing negative impacts on patients.

OBJECTIVE

To evaluate meaningful changes in clinical characteristics and outcomes among unstable angina (UA) patients admitted before and after the onset of the COVID pandemic.

METHODS

This is a single-center retrospective cohort study of consecutively admitted UA patients who underwent coronary angiography between January 2015 and April 2022. Two groups were defined according to the date of admission whether preceding or following March 2nd 2020: pre-COVID and post-COVID. Data on cardiovascular (CV) risk factors, medication, clinical history, echocardiography, and coronary angiography (CAG) were collected. Significant coronary artery disease (CAD) was defined as $\geq 70\%$ stenosis or a positive functional evaluation of a major epicardial vessel. One-year major cardiovascular adverse events (MACE) were defined as all-cause mortality, cardiovascular mortality, acute myocardial infarction, coronary revascularization, stent thrombosis, and stroke. Statistical analysis was performed using SPSS v28.

RESULTS

Overall, 166 patients were included in this study and 116 (70%) allocated to the pre-COVID group. Mean age was 64 ± 11 years, and the proportion of male patients was similar between the two groups (71% vs. 74%, $p=0.664$). There were no significant differences in terms of CV risk factors; however, the post-COVID group had a higher prescription rate of high-intensity statins (19% vs. 35%, $p=0.021$). There were no differences regarding significant CAD (51% vs. 48%, $p=0.735$), the number of diseased vessels (single vessel disease) (46% vs. 38%, $p=0.491$) and the time from pain onset to angiography (Time from pain onset to CAG $> 72h$) (41% vs. 54%, $p=0.134$). However, the post-COVID group presented a trend for higher rate of unsuccessful revascularization (9% vs. 27%, $p=0.073$). At discharge, there was a higher prescription rate of high-intensity statins plus ezetimibe (5% vs. 16%, $p=0.026$), but a lower prescription rate of ACE inhibitors (58% vs. 40%, $p=0.032$) in the post-COVID group. There were no significant differences in left ventricular ejection fraction (58% vs. 60%, $p=0.340$) and one-year MACE (6% vs. 4%, $p=0.722$).

CONCLUSIONS

The COVID pandemic represented a pivotal moment in modern medicine, requiring adjustments in hospitals to provide optimal care to patients. However, our study reveals that patients admitted for UA in the post-COVID group exhibited similar baseline characteristics, received comparable treatment, and did not present different outcomes.

P0 56

Genetic testing in hypertrophic cardiomyopathy: for all or just selected patients? A retrospective single-centre study

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Hypertrophic cardiomyopathy (HCM) is a genetic disorder characterized by left ventricular hypertrophy in the absence of abnormal loading conditions. Its prevalence is estimated at 0.16% to 0.29% in the general adult population and it is associated with important morbimortality such as heart failure, atrial fibrillation, stroke, and sudden cardiac death. Thousands of mutations in more than 60 genes have been described in association with HCM. MYH7 and MYBPC3, which encode the β -myosin heavy chain and myosin binding protein C, respectively, are the two most common genes involved, together accounting for about 50% of the HCM families. However, in approximately 40% of HCM patients, the causal genes remain to be identified. Some series have shown younger age, maximal wall thickness, or familial history as predictors of a positive genetic test. Thus, this study aims to identify clinical variables that might predict a positive genetic test result in HCM probands.

A retrospective single-centre study included all patients diagnosed with HCM for whom a genetic test was performed between 2019 and 2022. Patient selection was carried out by the Department of Clinical Pathology and information collection was obtained through medical records. Patients were divided into two groups: *Group 1* for patients with a negative genetic test, including negative

results and variants of uncertain significance; and *Group 2* for those with a positive genetic test, which includes the pathogenic/likely pathogenic variants. Group comparison tests were performed. A p-value less than 0.05 was statistically significant. Statistical analysis was performed using SPSS software v25.0.

Of the total of twenty-six patients with HCM, eighteen (69.2%) had a negative genetic test, and eight (30.8%) had a positive result. Six patients had a pathogenic mutation in the MYBPC3 gene, one patient in the Cysteine and Glycine Rich Protein 3 (CSRP3) gene, and another in the MYH6. Baseline characteristics (table 1) are similar between groups. The univariate analysis does not show significant differences between groups regarding the clinical presentation or results of initial tests (table 2).

In conclusion, we did not observe clinical predictors of a positive genetic diagnosis in our cohort, contrary to other series. These results reinforce the idea that genetic testing should be requested for all patients with HCM regardless of clinical findings, to refer them to genetic counseling whenever necessary. Finally, the authors recognize the small sample size as a limitation, so it would be important to carry out additional studies with large samples to clarify the results.

TABLE 1. BASELINE CHARACTERISTICS				
	Group 1 (N=18)	Group 2 (N=8)	Overall (N=26)	p-value
Male, n (%)	12 (66.7)	5 (62.5)	17 (65.4)	0.84
Age of onset in years, mean (dp)	54.2 (2.6)	61.7 (3.2)	56.5 (10.4)	0.11
Smoker, n (%)	3 (16.7)	0 (0.0)	3 (11.5)	0.53
Former smoker, n (%)	3 (16.7)	0 (0.0)	3 (11.5)	0.53
Hypertension, n (%)	11 (61.1)	6 (75.0)	17 (65.4)	0.49
Dyslipidemia, n (%)	7 (38.9)	6 (75.0)	13 (50.0)	0.09
Diabetes, n (%)	1 (5.6)	0 (0.0)	1 (3.8)	1.00
FH of HCM, n (%)	0 (0.0)	1 (12.5)	1 (4.0)	0.32
FH of SCD, n (%)	1 (5.9)	1 (12.5)	2 (8.0)	1.00

FH - Family history; HCM - Hypertrophic cardiomyopathy; SCD - Sudden cardiac death

TABLE 2. CLINICAL PRESENTATION AND RESULTS OF INITIAL EXAMS

	Group 1 (N=18)	Group 2 (N=8)	Overall (N=26)	p-value
Clinical presentation				
Chest pain, n (%)	2 (11.1)	1 (12.5)	3 (11.5)	1.00
Syncope, n (%)	0 (0.0)	1 (12.5)	1 (3.8)	0.31
Exertional dyspnoea, n (%)	2 (11.1)	1 (12.5)	3 (11.5)	1.00
Fatigue, n (%)	6 (33.3)	3 (37.5)	9 (34.6)	0.84
Systolic murmur, n (%)	5 (27.8)	4 (50.0)	9 (34.6)	0.27
Transthoracic echocardiogram (N=26)				
Left atrial diameter in mm, median (IQ)	46.0 (12.0)	40.0 (15.0)	45.5 (15.0)	0.28
LVEF in %, mean (dp)	62.2 (5.8)	59.9 (5.1)	61.3 (5.5)	0.40
MWT in mm, mean (dp)	19.9 (3.6)	19.1 (1.8)	19.7 (3.1)	0.57
SAM, n (%)	7 (38.9)	2 (25.0)	9 (34.6)	0.49
LVOTO, n (%)	6 (33.3)	1 (12.5)	7 (26.9)	0.27
MR (moderate or severe), n (%)	0 (0.0)	1 (12.5)	1 (3.8)	0.13
Cardiac magnetic resonance (N=20)				
LVEF in %, mean (dp)	69.9 (5.5)	66.3 (5.9)	68.9 (5.7)	0.20
LGE, n (%)	11 (78.6)	4 (66.7)	15 (75.0)	0.61
MWT in mm, mean (dp)	19.2 (6.1)	22.0 (2.9)	20.1 (5.4)	0.30
SAM, n (%)	6 (42.9)	3 (50.0)	9 (45.0)	0.77
Ventricular aneurysm, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	NA
Electrocardiogram (N=26)				
AF, n (%)	1 (5.6)	1 (12.5)	2 (7.7)	0.53
AVB 1st degree, n (%)	2 (11.1)	3 (37.5)	5 (19.2)	0.28
AVB 2nd degree Mobitz 1, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	NA
AVB 2nd degree Mobitz 2, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	NA
QRS complex duration in msec, mean (dp)	95.4 (40.3)	121.0 (22.3)	103.3 (37.3)	0.11
LBBB, n (%)	1 (5.6)	1 (12.5)	2 (7.7)	0.53
RBBB, n (%)	2 (11.1)	2 (25.0)	4 (15.4)	0.56
LVH criteria, n (%)	9 (50.0)	4 (50.0)	13 (50.0)	1.00
Inverted T waves, n (%)	7 (38.9)	2 (25.0)	9 (34.6)	0.49
24-Hour Holter Monitoring (N=25)				
AVB 1st degree, n (%)	3 (17.6)	2 (25.0)	5 (20.0)	1.00
AVB 2nd degree Mobitz 1, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	NA
AVB 2nd degree Mobitz 2, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	NA
NSVT, mean (dp)	3 (17.6)	2 (25.0)	5 (20.0)	1.00

LVEF - left ventricular ejection fraction; MWT - Maximal wall thickness; SAM - systolic anterior motion; LVOTO - left ventricular outflow tract obstruction; MR - mitral regurgitation; AF - atrial fibrillation; AVB - atrioventricular block; LBBB - left bundle branch block; RBBB - right bundle branch block; LVH - left ventricle hypertrophy; NSVT - non-sustained ventricular tachycardia

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