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EDITORIAL

Heart failure management: an endless journey

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Heart failure is a common clinical syndrome with important impacts in the healthcare systems. Newer pharmacotherapies have led to a decrease in hospitalizations, preservation of the quality of life and a decrease in mortality.

Digoxin, once the cornerstone of therapy for chronic heart failure, along with loop diuretics, is used rarely today. It may be considered only in patients with heart failure and reduced ejection fraction (HFrEF) and atrial fibrillation with rapid ventricular rate (rAF), not responding appropriately to guideline-directed medical therapy or who cannot utilize other therapies[1].

Following the digoxin-loop diuretic era, newer drugs, addressing the renin-angiotensin-aldosterone and the sympathetic systems, have been introduced, with favourable effects in both morbidity and cardiovascular mortality. These are, the angiotensin converting enzyme inhibitors (ACE-i) and the angiotensin receptor blockers (ARBs), the beta blockers (BB) and the mineralocorticoid receptor antagonists (MRAs), which have revolutionized the management of heart failure[2-5].

More recently, ivabradine[6] a selective sinus-node inhibitor, was related with fewer hospital admissions for worsening of symptoms and fewer deaths due to heart failure in patients with chronic heart failure.

In addition, another new medicine, sacubitril/valsartan[7], which combines a neprilysin inhibitor with an angiotensin receptor blocker, is recognized today as a first-line therapy for stable HFrEF patients, as well as in stabilized patients admitted with acute decompensation of heart failure.

Vericiguat, a novel oral soluble guanylate cyclase stimulator, has also been related with favourable outcomes, especially in reducing hospitalizations[8], whereas sodium-glucose transport protein-2 (SGLT2i) inhibitors, approved initially in 2013 to treat type-2 diabetes, have evolved, starting from 2018, as effective drugs initially for HFrEF and more recently across the whole spectrum of LVEF[9].

Cardiac Resynchronization Therapy (CRT) is recommended for symptomatic HFrEF patients, who are in sinus rhythm and have a QRS duration of ≥130 ms and preferably ≥150 ms, and left bundle branch block (LBBB) pattern[10].

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Patients at the higher end of heart failure severity, may benefit from heart transplant. With the limited number of heart donors however, it is more likely that most end stage HFrEF patients will be rather undergoing treatment with a left ventricular assist device (LVAD), not only as a bridge to transplant but also as a destination therapy. This may extend their life span, maintaining a relatively good quality of life[11].

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IMAGES IN CARDIOLOGY

Recurrent subvalvular aortic stenosis in a female patient, involving a two-level obstruction successfully treated surgically

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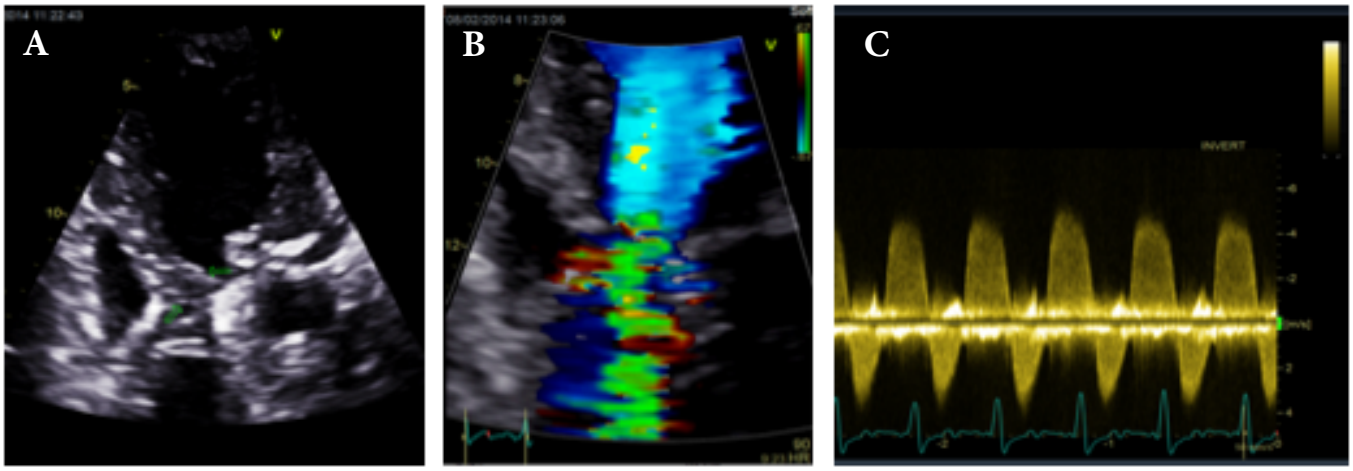
Recurrent subvalvular aortic stenosis in a female patient involving a rare two-level obstruction successfully treated surgically.

Subvalvular aortic stenosis (SAS) is a rare disorder with unclear etiology and variable clinical presentation, which predominantly involves males. It is caused by a discrete crescent-shaped membrane below the aortic valve (AV) or a thick fibromuscular ridge, and rarely due to a fibromuscular channel involving the whole left ventricular outflow tract (LVOT). SAS usually develops early in life with features of LVOT obstruction, left ventricular hypertrophy (LVH), and aortic regurgitation (AR) due to aortic valve destruction. While postoperative survival is excellent, most patients experience recurrent SAS.

We report the case of a 54–year-old woman, with medical history of atrial fibrillation and SAS surgery, who presented with dyspnoea. The transthoracic echocardiogram (TTE) showed normal dimensions and preserved systolic function of the LV. There was SAS at two discrete levels in the LVOT, a membrane immediately proximal to the AV and a fibromuscular bridge more distally, with a transmem-



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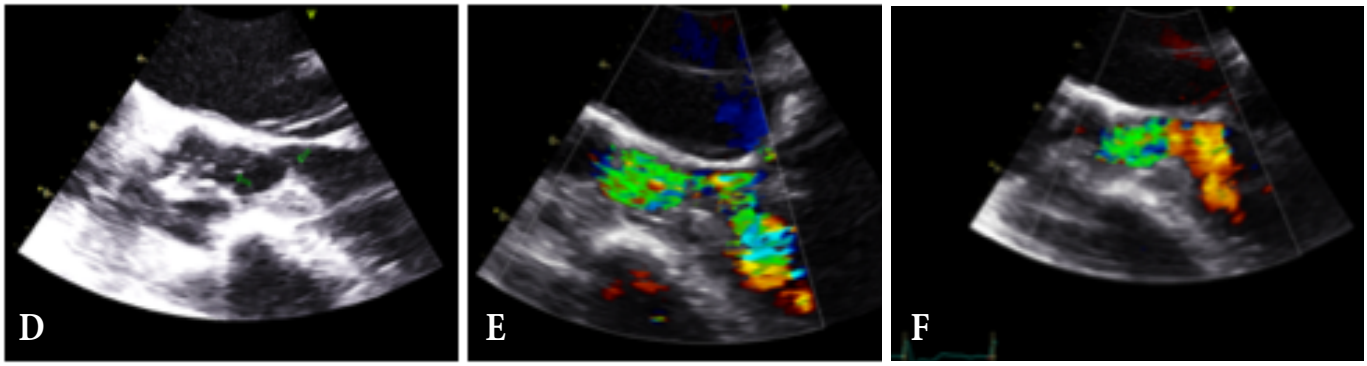
branous peak pressure gradient of 109 mmHg (pictures A, C). The AV was tricuspid, and AR was severe (picture B). The transesophageal echocardiogram confirmed the above-mentioned findings (picture D, E, F), and revealed an ascending aortic aneurysm. The patient was referred for surgery; Bentall procedure was performed, the subvalvular membranes were resected, and the LVOT was enlarged by septal myectomy. The postoperative TTE showed no flow acceleration in the LVOT, and a normal pressure gradient across the AV with no AR.

We present a unique case of recurrent SAS caused by a membrane and a fibromuscular bridge in a female patient successfully treated with redo surgery

Figure 1. 5-chamber transthoracic (TTE) and 5-chamber transesophageal (TEE) views demonstrating the presence of two discrete membranes in the LVOT (pictures A, D, green arrows), the distal one showing a gradual thickening and ending as a muscular ridge (picture D). Color Doppler recording of the same views (pictures B, E) showing a mosaic pattern of systolic flow along both membranes. 5-chamber TTE view with CW Doppler showing the high gradient flow across both membranes in systole and the presence of significant aortic regurgitation (AR) in diastole (picture C). 5-chamber TEE view with color Doppler in diastole (picture F) showing severe AR.



IMAGES IN CARDIOLOGY

Three-dimensional echocardiography views of the mitral valve

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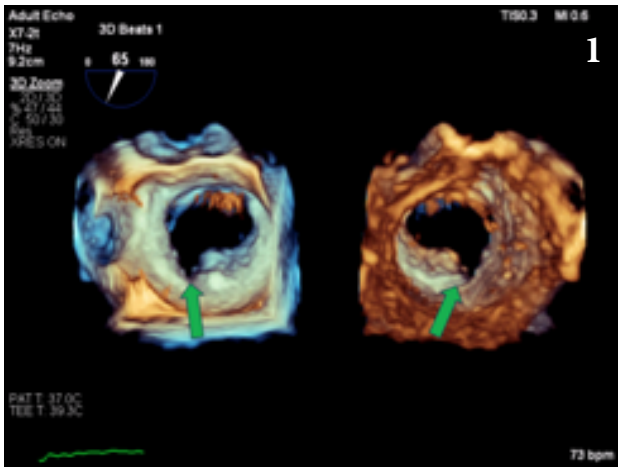
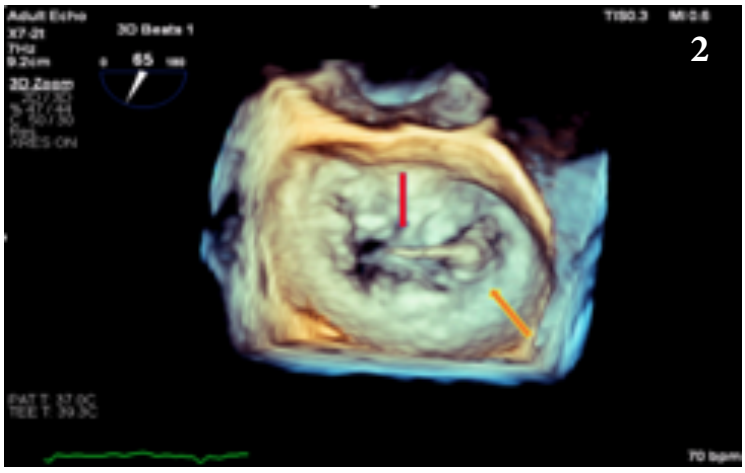
Three-dimensional (3D) echocardiography, has transformed the evaluation of mitral valve pathologies. The utilization of 3D echocardiography enables a more realistic visualization of the mitral valve, precise identification of lesions such as flailed leaflets, and clefts which can easily be missed on 2D echocardiography. The detailed anatomical insights and comprehensive assessments of the mitral valve offered by 3D echocardiography have revolutionized the management of mitral valve diseases, resulting in enhanced patient outcomes.

Image 1 shows a 3D image of the mitral valve during diastole. The valve is displayed with the aortic valve placed centrally and superiorly (12 o' clock). On the left, in the "en-face" or surgeon's view, we are viewing the valve from the atria, and on the right the valve is viewed from the ventricles.
The green arrows point to the cleft-like indentation of the posterior leaflet, between P1- P2. This will most probably need to be addressed by the surgeons during surgery.

Image 2 Surgeon's view of the same mitral valve during systole. The red arrow points to the ruptured chord while the orange arrow points to the P3 segment which is flail.





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REVIEW ARTICLE

What is new in hypertension guidelines 2023

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Abstract

The aim of our manuscript is to provide an overview of the major changes regarding the management of hypertension, comparing the new guidelines of 2023 with those of 2018. Moreover, we will discuss about the diagnosis, the treatment and our treatment targets and how we have to react under extreme circumstances.

Introduction

During the 32nd annual European meeting on Hypertension and Cardiovascular Protection which held in Milan, Italy in June 2023, the European Society of Hypertension (ESH) released updated and expanded 2023 ESH Guidelines for the management of arterial hypertension. Hypertension is a significant public health concern due to its widespread prevalence. Frequently, symptoms go unnoticed until a severe cardiovascular event, such as a stroke, heart attack, or chronic kidney disease, occurs. According to the new guidelines of 2023, our priority must be mainly given on early recognition and treatment of hypertension, in order to eliminate the risk of the aforementioned cardiovascular outcomes and overall mortality.

Discussion: Blood pressure thresholds and targets

Hypertension is defined based on repeated office Systolic Blood Pressure (SBP) values greater than 140 mmHg and/or Diastolic Blood Pressure (DBP) greater than 90 mmHg. Following the new guidelines, a greater number of individuals will be diagnosed with stage 1 hypertension, resulting in an increased need for non-drug interventions. However, patients with stage 1 hypertension and high cardiovascular disease (CVD) risk should receive blood pressure-lowering drug therapy, thus accurate estimation of clinical atherosclerotic CVD risk or estimated 10-year CVD risk becomes essential. Besides the precise CVD estimation, we have to observe, monitor and evaluate the function of all the target-organs. In comparison to 2018, Cardiac Magnetic Resonance Imaging (C-MRI) and Computer Tomography (CT) of the heart are the "new entries" in our list of our diagnostic tools. C-MRI is the gold standard for quantification of cardiac structure and function in clinical studies. In addition, arteries (carotid artery Intima Media Thickness (IMT)) and plaques (Pulse wave velocity, Ankle-Brachial Index), kidneys (reduced renal function, albuminuria), eyes (fundoscopy)

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and brain (MRI) should be regularly examined with the suitable tests. The new 2023 guidelines do not have major threshold changes in special groups. In patients 18-64 years old, the target for BP is <130/80mmHg. For 65-79 years old, the primary goal for BP is <140/90mmHg and if the treatment is well tolerated, lowering BP below 130/80mmHg may be considered. For those with Isolated Systolic Hypertension (ISH), the primary goal is to reduce SBP in the range of 140-150mmHg and if well tolerated, in the range of 130-139mmHg. For patients older than 80 years old, the goal for SBP is in the range of 140-150mmHg and DBP < 80mmHg. For frail patients, the target for office SBP and DBP must be individualized.

Treatment

In general, as we already know, all the patients diagnosed with high normal hypertension or greater, have to modify their lifestyle. The new guidelines emphasize the necessity of a healthy lifestyle in regulating blood pressure. Regarding drug treatment, there are no significant changes. There are five categories of drugs commonly used, single or combined, to effectively reduce BP and CV events. These categories are Angiotensin-Converting Enzyme inhibitors (ACEi), Angiotensin Receptor Blockers (ARBs), B blockers (BBs), Calcium Channel Blockers (CCBs) and diuretics, and are recommended as the basis of antihypertensive treatment strategies. Initiation of therapy with a two-drug combination is recommended for most of the patients and if BP is not controlled, a three-drug combination must be applied, preferably as a Single Pill Combination (SPC). According to Guideline Directed Medical Therapy (GDMT), BBs should be used as part of the initial therapy or at any treatment step when hypertension is accompanied by certain comorbidities such as Heart failure with reduced Ejection Fraction (HFrEF), anti-ischemic therapy in Chronic Coronary Syndromes (CCS), heart rate control in atrial fibrillation (AF). An important difference of these guidelines is the upgrading of BBs in the treatment algorithms, suggesting that BBs can be used at any step of the treatment algorithm if there is a guideline directed indication or other conditions for which they are thought to be beneficial. According to the new guidelines, in patients with HFrEF, alpha-1 blockers (doxazosin) can be used to prevent hypertension in combination with thiazide/thiazide-like diuretics and BBs in order to avoid fluid retention and tachycardia respectively. Sodium Glucose co-trasporter-2 inhibitors (SGLT-2is) are recommended for the prevention of heart failure in patients with type-II diabetes. If BP is not controlled by the four major drug classes and a diuretic, then a dihydropyridine (DHP)-CCB could be suggested. For patients with hypertension and heart failure with preserved ejection fraction (HFpEF), SGLT-2is should be used independently of the presence of type-II diabetes. Another group of hypertensive patients are those with chronic kidney disease (CKD). An ACEi or an ARB, titrated to the maximum tolerated doses is suggested for patients with CKD with moderate [urine Albumin to Creatinine Ratio (uACR) = 30 to 300 mg/g] or severe (uACR > 300 mg/g) albuminuria. SGLT-2is are suggested for patients with CKD, even estimated Glomerular Filtration Rate (eGFR) is at least 20 or 25 ml/min/1.73^2. The non-steroidal Mineralocorticoid Receptor Antagonist (MRA) finerenone, is suggested in

patients with CKD and albuminuria associated with type-II diabetes if eGFR is at least 25 ml/min/1.73² and serum potassium < 5.0 mmol/L. So, in summary one pill (if needed combination pill) is preferable, once daily (indifferent if morning or evening), aiming to the best compliance of our patient. If the patient has resistant hypertension or other comorbidities, then we have to adjust the antihypertensive therapy accordingly.

True Resistant Hypertension

True resistant hypertension is defined when SBP is ≥ 140mmHg or DBP is ≥ 90 mmHg provided that the maximum recommended and tolerated doses of a three-drug combination comprising a Renin-Angiotensin System (RAS) blocker (either an ACEi or an ARB), a CCB and a Thiazide/Thiazide-like diuretic were used, adequate BP control has been confirmed by Ambulatory Blood Pressure Monitoring (ABPM) and various causes of pseudo-resistant hypertension or secondary hypertension have been excluded. It is usually associated with Hypertension Mediated Organ Damage (HMOD) and increased CV risk. As additional therapy, preference is given in using spironolactone (or other MRAs), beta-blockers (BBs), alpha-1 blockers, amiloride or centrally acting agents. Thiazide/thiazide-like diuretics are recommended in resistant hypertension if eGFR is higher than 30 ml/min/1.73m2. Loop diuretics can be used if eGFR is lower than 45 ml/min/1.73m2 and should be used if eGFR is lower than 30 ml/min/1.73m2. Renal denervation (RDN) is considered as an additional treatment option in patients with an eGFR greater than 40 ml/min/1.73m2. Patients with resistant hypertension require close monitoring.

Implementation

Monitoring adherence to BP guidelines across Europe constitutes a challenge, due to the absence of a standardized population survey for each nation, as required by the guidelines committee. An imperative need arises for a European population survey, executed uniformly based on similar implementation methods, aiming the more accurately track of hypertension prevalence and its control rates. Recent data, indicates a fluctuation in hypertension prevalence across countries, but it remains generally high within Europe and globally. In order to enhance the implementation process, a comprehensive proposed model should include several crucial facets. First and foremost, the model should focus on utilizing methods that ensure accurate and precise measurement of BP. The incorporation of tracking systems to monitor the patient's daily progress, seems to result in better BP control for adults with uncomplicated hypertension. Moreover, health promotion through national strategies would be essential, facilitated by easy accessibility to healthcare teams (given that the healthcare providers should be aware about established guidelines) and availability of antihypertensive medications (with a preference for single-pill combinations). Finally, the adoption of a case-management approach is considered very important, leading not only to a precise practise, but also to an effective implementation of BP guidelines.

Conclusion

Comparing the guidelines of 2018 with the new recommendations of 2023, we realize that hypertension diagnosis and treatment approaches, generally remain mostly the same.

Only limited practices have undergone changes. Randomized Controlled Trials (RCTs), despite their limitations and biases, are essential in providing evidence to medical societies. In the upcoming years, hopefully, we will be able to "fill the gaps" and apply evidence-based and tailored treatments for our patients. Machine learning and artificial intelligence may play a crucial role in achieving this task. We should never forget that each patient is unique, so the approach and evaluation must be individualized, to provide high-quality health services.

Final Remarks

The management of hypertension is crucial. The patients should be usually observed and their medication should be re-evaluated and altered if the therapy-target is not achieved. New exams are nowadays used to estimate CVD risk and according to their results and patients' co-morbidities, the patients are categorised in different groups. As a result, their medication may be updated as well. The treatment-options of hypertension remain mainly the same, but new therapy strategies are under investigation. Data bases and clinical trials are to have an important role for our society to make the next step in the management of hypertension.

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3. Cardiovascular magnetic resonance in the guidelines of the European Society of Cardiology: a comprehensive summary and update (Florian von Knobelsdorff-Brenkenhoff and Jeanette Schulz-Menger)

CLINICAL ARTICLE

Bronchogenic Takotsubo Syndrome: A Case Report

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Abstract

Bronchogenic stress cardiomyopathy, a rare and often insufficiently recognized subset of Takotsubo cardiomyopathy (TCM), has been documented to manifest concurrently with acute exacerbations of Chronic Obstructive Pulmonary Disease (COPD). Within this context, we present a case of a 73-year-old male who was admitted to our hospital due to an acute COPD exacerbation and received conventional high-dose bronchodilator therapy as part of his treatment regimen. Subsequently, he exhibited clinical indicators closely mirroring those of acute coronary syndrome. However, an urgent coronary angiogram unveiled unobstructed coronary arteries prompting a potential diagnosis of Takotsubo Cardiomyopathy. This diagnostic assessment was substantiated by the complete restoration of cardiac function to baseline levels, observed upon the patient's discharge. Notably, this clinical case marks the patient's second episode of TCM within five years, with the exacerbation of COPD once again identified as the triggering factor.

Keywords: Case Report, Chronic Obstructive Pulmonary Disease (COPD), Takotsubo Syndrome (TTS), Stress - cardiomyopathy, Beta-agonist

Introduction

Takotsubo Syndrome (TTS), also known as transient apical ballooning syndrome, involves reversible decreased systolic function, triggered by either a physical or emotional stressor, and importantly, it occurs without any indication of coronary artery disease (CAD)[1]. TTS predominantly afflicts postmenopausal women, as documented in the medical literature [2]. This clinical condition emerges within an estimated 1-2% of cases where individuals present with clinical manifestations suggestive of acute coronary syndrome [3].

"Bronchogenic Takotsubo Syndrome" has been introduced as a particular form of TTS that emerges in the setting of severe acute dyspnea in asthma or COPD, with atypical presentation [11]. According to the current literature, the observed prevalence of concurrent COPD in individuals diagnosed with Takotsubo cardiomyopathy (TCM) appears to be approximately 21.8% [4]. This brief article introduces the concept of "Bronchogenic Takotsubo Syndrome", setting the stage for a deeper dive into its clin-

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ical relevance and potential implications for patient management. In this report, we present a compelling case of a 73-year-old male who was admitted to our hospital due to an acute COPD exacerbation and subsequently manifested signs and symptoms consistent with Takotsubo cardiomyopathy, marking his second occurrence within the past five years.

Case presentation

A 73-year-old male presented to the emergency department (ED) with a gradually worsening dyspnea that had developed since the previous night. There was no associated cough, fever, or other flu-like symptoms. The patient's medical history included a diagnosis of COPD and a significant smoking history of 60 pack-years. He had been using inhalation bronchodilators regularly at home for COPD management. Interestingly, despite his lack of awareness regarding any preexisting heart conditions, he was under the care of a cardiologist and his medication regimen encompassed diuretics – Furosemide 40 mg once daily, Spironolactone 25 mg once daily - as well ACEi (Ramipril 5 mg) and a beta-blocker (Carvedilol 6.25 mg twice daily). He also reported that he had undergone a coronary angiogram 5 years prior, which did not necessitate any coronary intervention. Upon examination, the patient exhibited no fever, and his blood pressure was measured at 103/75 mmHg. However, his oxygen saturation level (SatO2) was notably low, measuring at 85% on room air, and his heart rate (HR) was 78 beats per minute. The cardiovascular examination yielded unremarkable findings, while the respiratory examination revealed bilateral wheezing. The arterial blood gas analysis unveiled a state of chronic respiratory acidosis, as evidenced by a pH level of 7.3, a PaCO2 reading of 55 mmHg, a PaO2 measurement of 62 mmHg, and a bicarbonate (HCO3) concentration of 27 mmol/L. Chest x-ray demonstrated typical findings of emphysema with lung hyperinflation, flattened hemidiaphragmsand also signs of chronic bronchitis with increased bronchovascular markings.

Taking into account the patient's clinical presentation and documented history of COPD, a distinct episode of acute exacerbation in his re-

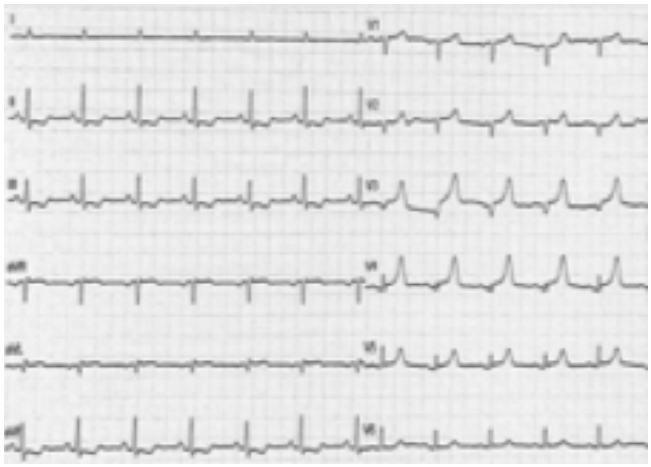


Figure 1A. *Resting 12-lead electrocardiogram demonstrated sinus rhythm with ST-segment elevation in V1-5, with reciprocal ST- segment depression in II, III, a VF.*

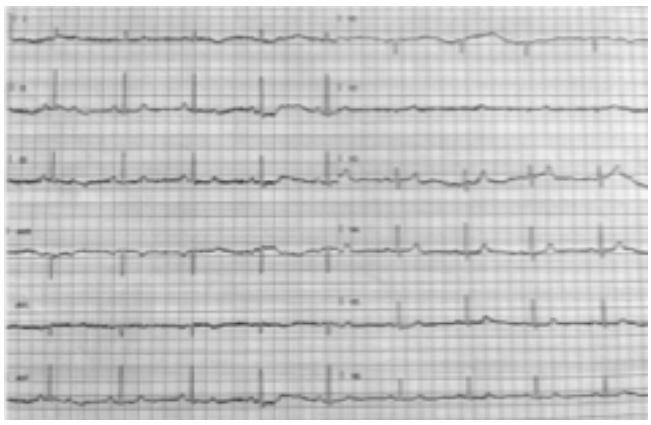


Figure 1B. *Resting 12-lead electrocardiogram demonstrated sinus rhythm, without ST-T segment changes.*

spiratory status becomes evident. Subsequently, therapeutic interventions were initiated, involving nebulized bronchodilators, encompassing a beta-2 agonist (Ventolin) and ipratropium bromide (Atrovent). Further, in conjunction with a supplementary oxygen supply delivered via nasal cannula at a rate of 3 liters/minute, the patient was administered a systemic corticosteroid, 125 mg of Solumedrol.

Notably he didn't complain of any chest pain, however his resting electrocardiogram (ECG) findings revealed sinus rhythm SR, accompanied with Q waves in V1-3 , coupled with ST – segment elevation in V1-5 , with reciprocal ST-segment depression in II, III and aVF *(FIGURE 1A).* These alterations were newly observed in contrast to his prior electrocardiogram (ECG) recorded one year ago. *(FIGURE 1B).*

Additionally, elevated cardiac biomarkers suggestive of myocardial damage were observed, with a troponin-I level of 2.46 ng/ml (normal range <0.5ng/ml) and with a CK-MB of 65 IU/L (normal range < 25 IU/L).

Bedside transthoracic echocardiography (TTE) revealed severe hypokinesia of mid and apical anteroseptal wall and severe hypokinesia of apex, with no evident thrombus. Left ventricular systolic function was moderately impaired, with an estimated ejection fraction (EF) 30-35%). RV was normal in size and systolic function. No significant valvulopathies and no pericardial effusion was observed. Proximal ascending aorta was within normal limits. Upon a brief inquiry to his medical electronic records, we retrieved a prior assessment by his cardiologist and his transthoracic echocardiogram (TTE) a year ago, indicated a satisfactory left ventricular systolic function with an ejection fraction (EF) within the range of 50-55%.

Diagnosis

Subsequent to the TTE, the patient underwent an expeditious coronary angiogram procedure, followed by admission to the Cardiac Care Unit. Coronary angiography revealed atheromatous coronaries, free of significant disease with no changes in comparison with the coronary angiography in 2017 *(FIGURE 2).* The Left Ventriculography unveiled a severely impaired LV systolic function with apical anterior and apex and apical inferior hypokinesis, indicative of apical ballooning syndrome. It is noteworthy to highlight that back in 2017 the Left ventriculography

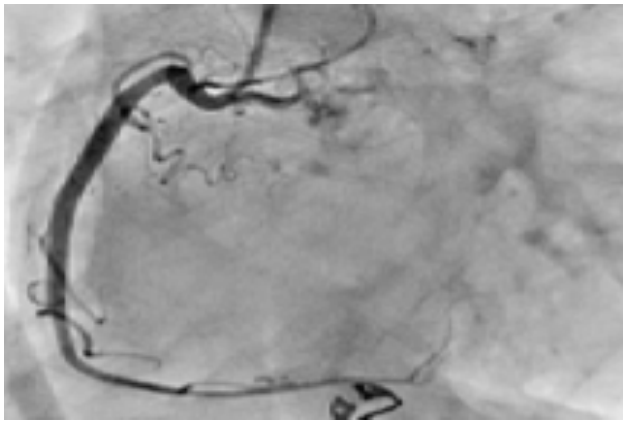
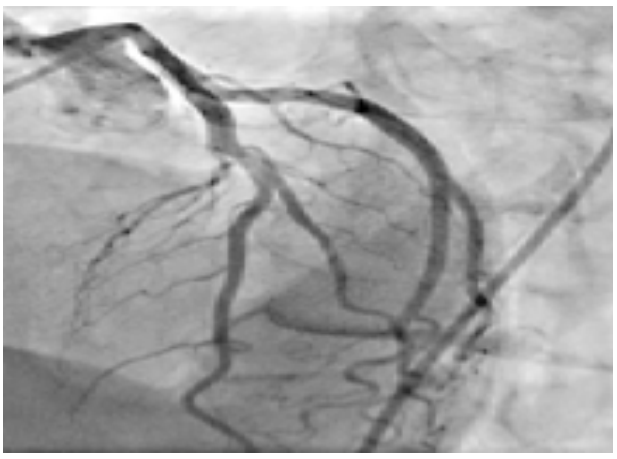


Figure 4. *Coronary angiogram revealed normal coronaries, free of significant disease.*
(2A) LAO view of RCA(2B) LAO cranial view of LCA



also demonstrated severe global hypokinesia and the diagnosis of Non – Ischemic Cardiomyopathy was conducted.

Based on a comprehensive evaluation incorporating the patient's medical history, the clinical syndrome closely resembling myocardial infarction, and angiographic results, a potential diagnosis of a recurrent episode of Takotsubo Cardiomyopathy was made. This occurrence was closely linked to a plausible trigger, conceivably attributed to the acute exacerbation of chronic obstructive pulmonary disease (COPD). In this particular case we had to deal with a recurrent episode of TTS, considering that the patient had previously experienced the same ailment back in 2017. Significantly, the transthoracic echocardiogram (TTE) conducted during the patient's discharge exhibited a restoration of the left ventricle's systolic function to a generally normal state. This observed reversibility confirms our diagnosis of Takotsubo Cardiomyopathy.

Treatment

The patient's clinical condition improved the next days, as evidenced by the alleviation of dyspnea and his condition remained stabilized throughout his hospitalization. The therapeutic approach encompassed the systemic administration of inhaled bronchodilators, specifically ipratropium bromide, as well as intravenous corticosteroids, accompanied by the discontinuation of β2-agonists. His existing regimen was maintained, comprising Carvedilol, Aldactone, and Ramipril, alongside a conservative dosage of 20 mg of Furosemide administered daily.

Discussion

Takotsubo Syndrome (TTS) stands as a complex and enigmatic medical condition, characterized by a characterized by a pathophysiology that remains notably multifactorial and inadequately understood.The genesis of TTS frequently intersects with emotional or physical stressors, with the sympathetic stimulation being as a pivotal factor [12].It has been proposed that epinephrine has a stunning effect on myocardial tissue in TTS due to the induction of beta-2 receptors, which are more frequently found in the cardiac apex [8]. Within this context, the pathogenesis of Bronchogenic TTS entails a mechanism that involves myocardial stunning induced by excessive catecholamine release, which dominates during acute COPD exacerbation. [5]. It is noteworthy, that elevated catecholamine concentrations have been observed in COPD patients, particularly during the administration of high-dosage of beta-agonists (such as salbutamol) for managing acute exacerbations. Consequently, the prospective advantages of administering beta-blockers could be applicable to patients experiencing Takotsubo cardiomyopathy (TCM) secondary to acute COPD [6]. Nevertheless, the utilization of beta blockers in the context of Takotsubo Syndrome (TTS) remains a subject of debate, and existing data indicate a lack of discernible survival advantages associated with beta blocker administration within a one-year timeframe [9]. Regarding to the utilization of ipratropium, its application appears to have favorable effects in the context of "bronchogenic TTS", as it holds the potential to mitigate the necessity for beta-2-agonist administration [11].

Moreover, the stressors that predominate during episodes of acute exacerbation of chronic obstructive pulmonary disease (COPD), such as hypoxia and acidosis, possess the capacity to incite activation of cardiac beta-2-ad-

renergic receptors, thereby contributing to the potential pathogenesis of TCM [7].

In addition, given its status as a chronic systemic inflammatory ailment, COPD can induce an elevated IL-6 level, a phenomenon that could potentially accentuate the inflammatory cascade and contribute to the pathogenesis of the disease [13].

Bronchogenic Takotsubo cardiomyopathy (TCM), as an atypical form, manifests in a subtle manner, predominantly marked by the symptom of dyspnea, notably devoid of accompanying chest pain [14]. Thus, the diagnosis of TTS during acute exacerbations of COPD can be challenging. In this context, the significance of performing repetitive electrocardiogram (ECG) assessments becomes evident, serving to distinguish between prolonged, uncomplicated bronchospasm and bronchogenic stress cardiomyopathy. Consequently, the atypical presentation of TTS can mask underlying cardiac issues, delay prompt TTS diagnosis, and allow unchecked disease progression, leading to poor outcomes.

According to available data, individuals with COPD and concurrent TTS experience unfavorable in-hospital outcomes. A retrospective cohort study conducted by Li P et al, involving 3139 patients with TTS, underscored a distinct pattern within patients with concurrent COPD.Within this subset, a discernibly higher prevalence of acute respiratory failure (22.6% vs. 8.2%, $P < 0.001$), cardiogenic shock (5.6% vs. 3.3%, $P = 0.024$), and in-hospital mortality (2.9% vs. 1.0%, $P = 0.005$) was observed. Notably, individuals with COPD also exhibited elevated hospitalization expenses and prolonged durations of stay, in comparison to their patients without COPD [4].

Conclusion

"Bronchogenic TTS" although rare, presents an ongoing challenge in the landscape of clinical practice, necessitating timely identification and diagnosis for appropriate therapeutic interventions. In light of its atypical presentation, where the absence of chest pain is noteworthy, clinicians are advised to ask for repeated electrocardiograms (ECGs) and if abnormalities observed, an early, point-of-care echocardiogram stands as a pivotal initial step towards the diagnosis. In such cases, the use of a beta-2 agonist should be limited or avoided and other treatment options, such as inhaled ipratropium and corticosteroids, should be prioritized.

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The European Exam in Core Cardiology

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

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

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

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

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

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

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

The following information is necessary for registration:

- Email address
- ID number
- Phone number

Candidates receive a confirmation e-mail from the ESC.

Exam preparation

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

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

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

The following modules are part of the EECC.

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Module 2. Coronary artery disease
Module 3. Valvular heart disease
Module 4. Rhythm disorders
Module 5. Heart Failure
Module 6. Acute Cardiovascular Care
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A Certificate is issued to the candidates, who pass the Exam, by the Cyprus Society of Cardiology in collaboration with the ESC and the UEMS.

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