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# ORAL PRESENTATIONS



## [OP-01]

### A case of complex interplay between heart failure, severe aortic stenosis and severe renal impairment

Melike Demir, Semih Kalkan, Alev Kılıçgedik, Mehmet Rasih Sonsöz

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Renal artery stenosis is an important reason of renal function impairment and can lead to worsening of heart failure and increased mortality. Whether renal artery revascularization is needed before transcatheter aortic valve implantation (TAVI), is unknown. Herein, we present an 82-year-old woman who was admitted with progressive dyspnea lasting four days. Her medical history was significant for prior coronary artery bypass graft, hypertension, diabetes mellitus and chronic kidney disease. On admission BP was 170/90 mmHg, and the patient was desaturated on room air. Physical examination revealed tachypnea and bilateral pretibial edema. Electrocardiogram showed atrial fibrillation without significant ST-T changes. Chest X-ray revealed cardiomegaly and bilateral pleural effusion. Her creatinine was elevated (2.5 mg/dL), epidermal growth factor receptor (eGFR) was reduced (16 mL/min/1.73 $m^2$) and NT-proBNP was elevated (23,500 Pg/mL). TTE showed LVEF 48%, severe aortic stenosis (AVA 0.85 $cm^2$), pulmonary hypertension (estimated systolic pulmonary artery pressure 55 mmHg). Despite administration of furosemide 480 mg/day, daily urine output remained below 3 liters. Oral acetazolamide (250 mg twice daily) was added, however effective diuresis could not be achieved. A right-sided thoracic drainage tube was inserted for pleural effusion, and repeated ultrafiltration sessions were performed. Despite these measures, the patient continued to exhibit refractory congestion and uncontrolled hypertension. Abdominal computed tomography demonstrated bilateral ostial renal artery calcifications suspicious for renal artery stenosis. Both kidneys measured approximately 9 cm longitudinally, suggesting preserved viability. Invasive renal angiography revealed 95% stenosis of the right distal renal artery, 80% stenosis of the left proximal renal artery. A 6×14 mm stent was successfully implanted in the left renal artery. The right renal artery lesion was deemed unsuitable for intervention. Following the procedure, the patient continued to receive intravenous furosemide and intermittent ultrafiltration. Although congestive symptoms persisted, blood pressure was successfully controlled (systolic <140 mmHg). Approximately two months after admission, she underwent TAVI. Within three days of the procedure, her congestive findings resolved completely, and she was discharged. At one-month outpatient follow-up, she was asymptomatic. TTE demonstrated a normally functioning bioprosthetic aortic valve, and renal function remained stable (eGFR 20 mL/min/1.73 $m^2$). This case highlights the complex interplay between severe aortic stenosis, chronic kidney disease, and bilateral renal artery stenosis in a patient presenting with refractory heart failure. Left renal artery revascularization successfully stabilized blood pressure but did not fully resolve congestion. Definitive relief of heart failure symptoms was achieved only after TAVI. We think that a staged strategy combining renal artery revascularization and TAVI provided an optimal hemodynamic and clinical outcome in this patient.

**Keywords:** Cardiac failure, chronic renal insufficiency, renal angiography, transcatheter aortic valve implantation.

## [OP-02]

### A diagnostic dilemma: Athlete's heart or cardiomyopathy?

Güldane Nevra Güner, Mehmet Serkan Çetin

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**Background:** Athlete's heart is a physiological condition associated with the changes that occur in people that perform regular and long term physical activity. These changes may include increased mass, cavity dimensions and wall thickness with normal systolic and diastolic functions. The causes of these changes are not fully known but they depend on the type and duration of the activity. Sometimes these adaptive changes in an athlete's heart may overlap with pathological conditions such as cardiomyopathies that are associated with an increased risk of sudden cardiac death. For this reason screening of individuals before competitive sports activities is recommended. In this case report, we present a young male military school student in the grey zone between athlete's heart and left ventricular non-compaction cardiomyopathy (LVNC) to highlight the importance of the differential diagnosis.

**Case Report:** A 22-year-old male patient with no known medical conditions and a five-year history of endurance training, predominantly running two to three days per week, was referred to our cardiology department for cardiovascular evaluation prior to military training. His physical examination and 12-lead electrocardiogram were unremarkable. He had no family history of sudden cardiac death. His echocardiography showed normal systolic wall motions and right ventricular function but slightly enlarged end-diastolic diameter (6.0 cm), near normal left ventricular ejection fraction (52.8%) and minor increase in LV trabeculation warranting further evaluation. Global longitudinal strain analysis was made using the Philips Epiq 7C aCMQ software to reveal a near normal value of -18.1%. Cardiac magnetic resonance imaging (CMR) demonstrated a mildly increased left ventricular end-diastolic volume of 197 mL and an end-systolic volume of 93 mL with a stroke volume of 104 mL and a cardiac output of 7.7 L/min. Prominent left ventricular trabeculation was also observed; however the overall findings did not meet the diagnostic criteria for LVNC. Cardiopulmonary exercise testing (CPET) demonstrated a peak oxygen uptake within the normal range. Oxygen pulse increased progressively throughout exercise and continued to rise until peak exercise, indicating an appropriate increase in stroke volume. Ventilatory efficiency was normal with no evidence of impaired oxygen delivery. Overall the CPET findings supported physiological cardiac adaptation to endurance training. In conjunction with the CMR findings these results favored athlete's heart rather than an underlying cardiomyopathy and the patient was cleared for military training.

**Discussion:** Athlete's heart is a condition associated with adaptations in a person who performs regular exercise. These changes are also observed in some cardiomyopathies. Our case demonstrates the significance of pre-participation screening. Accurate differentiation of athlete's heart from pathological conditions prevents missed diagnoses and unnecessary exclusion from sports.

**Conclusion:** This case emphasises the importance of careful cardiovascular evaluation of young individuals before participating in intensive physical training. Exercise-induced cardiac remodeling may mimic structural features of cardiomyopathy. Therefore a comprehensive assessment is required to distinguish physiological adaptation from pathological conditions.

**Keywords:** Athlete's heart, cardiomyopathies, cardiopulmonary exercise testing.

## [OP-03]

### Acute heart failure as the presenting feature of a silent myocardial infarction with ventricular septal rupture

Rohat Çiçek, Saadet Aydın, Zeynep Yüksel, İlhan Koyuncu, Ömer Faruk Rahman

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**Background:** Ventricular septal rupture (VSR) is a rare but highly lethal mechanical complication of myocardial infarction. Although its incidence has decreased with the widespread use of reperfusion therapies, it can still occur late in the course of untreated or undiagnosed myocardial infarctions. Silent myocardial infarctions, particularly in patients with risk factors, can lead to delayed diagnosis and the development of serious complications.

**Case Report:** A 63-year-old male patient with a history of hypertension, active smoking, and a prior ischemic cerebrovascular event presented to the emergency department with orthopnea and progressive dyspnea (NYHA class III-IV). He had no chest pain at presentation; however, history-taking revealed that he had experienced a severe, typical anginal episode approximately two months earlier and had not sought medical attention at that time. On physical examination, the patient was borderline hypotensive (94/58 mmHg), tachycardic ($10^9$/min), and hypoxemic ($SpO_2$ 80%, improving to 93% with supplemental oxygen). ECG showed sinus rhythm with complete right bundle branch block, along with aneurysmal ST-segment elevation in the precordial leads. Transthoracic echocardiography revealed an LVEF of 40%, with advanced hypokinesia of the anterior and apical septum, and a large muscular ventricular septal defect approximately 25 mm in diameter with a left-to-right shunt, localized within an aneurysmal septo-apical segment. Coronary angiography demonstrated multivessel coronary artery disease: 70% stenosis in the proximal LAD, total occlusion of the mid-segment and distal diagonal branch, and 80% stenosis in the distal RCA. The LAD was noted to be filled via collateral circulation, and chronic total occlusion was considered for both the LAD and the diagonal artery. Laboratory findings showed a mild elevation in troponin levels and a marked increase in NT-proBNP to 11,532 pg/mL (reference <125 pg/mL). The patient underwent surgery due to hemodynamic instability. Under cardiopulmonary bypass, the VSD was repaired with a pericardial patch; the aneurysmal segment was resected and closed, and bypass grafting to the right coronary artery was performed. The patient left the operating room in stable condition. A follow-up postoperative chest X-ray (PA view) obtained on postoperative day 10 showed marked improvement in pulmonary congestion and pleural effusion compared to the earlier study. The patient remained stable and was discharged on postoperative day 12.

**Conclusion:** This case demonstrates that myocardial infarction does not always present with typical symptoms, and that in untreated cases, life-threatening mechanical complications can develop at a later stage. In patients presenting with acute heart failure, particularly when risk factors and atypical electrocardiographic findings are present, VSR must always be kept in mind. Early echocardiographic evaluation is of critical importance for both diagnosis and treatment.

**Keywords:** Ventricular septal rupture, myocardial infarction, mechanical complication, silent myocardial infarction, cardiac surgery.

## [OP-04]

### Amyloidosis masked by GI symptoms: Diagnosis of cardiac and renal involvement with a multidisciplinary approach

Berna Akdeniz, Fatıma Aksoy, Bahri Akdeniz

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Amyloidosis is a heterogeneous group of disorders characterized by the extracellular deposition of misfolded proteins with a beta-pleated sheet structure. These deposits—which stain with Congo Red and exhibit apple-green birefringence under polarized light—can manifest with widely varying clinical presentations depending on the affected organ. Cardiac amyloidosis is an infiltrative cardiomyopathy that leads to restrictive physiology and can mimic hypertrophic cardiomyopathy on echocardiography through symmetrical or asymmetrical wall thickening. The most valuable clue in making this distinction is the observation of low voltage and/or a pseudo-infarct pattern on the electrocardiogram despite the increased wall thickness; this "echocardiography-ECG discordance" prompts consideration of an infiltrative process. The clinical presentation of amyloidosis is generally non-specific; signs of autonomic dysfunction—such as hypotension, dizziness, nausea, and syncope—are the initial presentation symptoms in a significant proportion of patients and are frequently misdiagnosed as vestibular pathologies or simple hypotension due to volume depletion, leading to delayed diagnosis. Literature indicates that the time elapsed before a diagnosis of AL amyloidosis is established can take months or even years, with patients receiving a diagnosis only after consulting multiple physicians or clinics. This diagnostic delay directly adversely affects prognosis, particularly in patients with cardiac involvement, as the restrictive physiology and advanced heart failure resulting from progressive amyloid infiltration can become irreversible. A 69-year-old female patient presented to an ED with complaints of recurrent dizziness, nausea, and vomiting. The patient had made numerous emergency department visits with similar complaints before. The patient was admitted to the cardiology service for the investigation of amyloidosis due to the discrepancy between echocardiographic hypertrophy and ECG low voltage. 7 grams of proteinuria were detected in a 24-hour urine sample. Cardiac MRI revealed asymmetric myocardial thickening—predominantly involving the interventricular septum, with relative sparing of the lateral wall (septal end-diastolic thickness: 17 mm). Late-phase contrast-enhanced images demonstrated diffuse subendocardial concentric late gadolinium enhancement and elevated T1 mapping values, particularly in the mid and apical anterior walls. Taken together, these findings were concluded to support a restrictive pattern of involvement consistent with amyloidosis. Amyloidosis was histopathologically confirmed via an abdominal skin incisional biopsy. Findings were consistent with the lambda-predominant monotypic plasmacytosis identified in the bone marrow biopsy, supporting the diagnosis of an underlying clonal plasma cell disorder and AL-type amyloidosis. This case report presents a patient who was monitored for a prolonged period with a diagnosis of peripheral vertigo and recurrent orthostatic symptoms, but was referred for further evaluation after inconsistencies were seen in echocardiographic and electrocardiographic findings; the patient was ultimately diagnosed with systemic AL amyloidosis involving the heart and kidneys. The case is presented through the collaborative assessment of the cardiology, nephrology, and hematology departments, and the diagnostic steps and treatment approach are discussed in light of current literature.

**Keywords:** Amyloidosis, cardiomyopathy, cardiac MRI, heart failure, proteinuria.

## [OP-05]

### Lead-induced tricuspid regurgitation and ascites in hypertrophic cardiomyopathy: Post-shock trauma

Fatıma Aksoy, Berna Akdeniz, Ebru Özpelit

*Division of Cardiology, Department of Internal Medicine, Dokuz Eylül University Hospital, İzmir*

**Background:** Tricuspid regurgitation (TR) secondary to permanent pacemaker and implantable cardioverter-defibrillator (ICD) leads is a well-recognized yet frequently underdiagnosed long-term complication that may progress to advanced right ventricular failure. While predominantly driven by leaflet impingement, TR severity is further compounded by right ventricle remodeling, tricuspid annular dilation, and pacing-induced cardiomyopathy. We report a case of severe lead-related TR manifesting as massive ascites thirteen years after ICD implantation for hypertrophic cardiomyopathy (HCM).

**Case Report:** A 48-year-old female presented with massive ascites, severe lower-extremity edema, and NYHA Class III-IV dyspnea. Her medical history revealed that a single-chamber (VVI) ICD had been implanted in 2012 for primary prevention of sudden cardiac death (SCD) following the diagnosis of HCM with a restrictive phenotype (interventricular septum thickness: 32 mm, left ventricular outflow tract gradient: 35 mmHg, and documented non-sustained ventricular tachycardia), in conjunction with a family history of premature SCD (mother at age 33, sister at age 16). During her pregnancy in 2015, she experienced 15 inappropriate shocks triggered by rapid atrial fibrillation, followed by an ischemic stroke in 2016. Due to profound psychological trauma induced by the recurrent inappropriate shocks during pregnancy, the patient refused battery replacement in 2017 and subsequently remained completely lost to clinical follow-up until the current presentation. Transthoracic echocardiography performed revealed preserved left ventricular systolic function, notable septal flattening secondary to right ventricular pressure overload, mid-apical cavity obliteration, and restrictive

physiology. Severe right-sided heart chamber enlargement was observed along with severe TR attributed to ICD lead adherence to the tricuspid leaflets. The massive ascites and peripheral edema were deemed secondary to lead-related right heart failure rather than the natural restrictive progression of HCM. The patient was stabilized with standard heart failure therapy, including diuretic treatment and close volume monitoring. Management options were evaluated via a multidisciplinary "Heart Team" approach comprising cardiology, electrophysiology, and cardiovascular surgery. Based on the consensus reached by the joint cardiology and cardiovascular surgery council, the patient underwent surgical tricuspid valve replacement and surgical lead extraction.

**Conclusion:** Lead-related severe TR, which is more frequent in hypertrophic cardiomyopathy with right ventricular involvement, may clinically mimic or compound underlying restrictive pathophysiology, leading to diagnostic oversight. In device recipients presenting with signs of right-sided heart failure, meticulous echocardiographic evaluation of lead position and tricuspid leaflet motion is imperative. Furthermore, this case underscores how psychological trauma from inappropriate shocks can impair treatment adherence, allowing a treatable complication to progress to end-stage right heart failure. Technical success translates into genuine clinical efficacy only when coupled with psychological resilience and sustained follow-up.

**Keywords:** False shock, hypertrophic cardiomyopathy, implantable cardioverter-defibrillator, lead-related tricuspid regurgitation, right heart failure.

## [OP-06]

### Cryptogenic stroke revealing late-onset Fabry disease through multimodality cardiac imaging

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**Background:** Fabry disease is an X-linked lysosomal storage disease caused by deficient α-galactosidase A enzyme activity and may present with cardiac, renal, and cerebrovascular involvement. Particularly in female patients, enzyme activity may be normal or near-normal, which can lead to diagnostic delay. This case report describes a female patient evaluated for cryptogenic stroke, in whom the diagnosis of Fabry disease was reached through multimodal cardiac imaging findings.

**Case Report:** A 58-year-old woman with hypertension, chronic kidney disease, and a history of cerebrovascular events was admitted to the neurology clinic with acute ischemic stroke. A cardiology consultation was requested as part of the etiological work-up. The patient had long-standing exertional dyspnea and reduced exercise capacity; her family history included end-stage renal disease requiring dialysis in her mother and maternal aunt. Electrocardiography showed sinus rhythm and signs of left ventricular hypertrophy. Creatinine was 2.3 mg/dL, BNP 680 pg/mL, and high-sensitivity troponin 23 ng/L. Transthoracic echocardiography showed preserved left ventricular ejection fraction with concentric left ventricular hypertrophy, stage 3 diastolic dysfunction, mild thickening of the aortic and mitral valves, and a marked reduction in global longitudinal strain, particularly in the basal inferolateral segments. Owing to suspicion of infiltrative cardiomyopathy, cardiac magnetic resonance imaging was planned for tissue characterization. Cardiac MRI revealed increased left ventricular wall thickness, low native T1 values, and patchy mid-myocardial late gadolinium enhancement in the inferolateral mid-basal segments. Unexplained hypertrophy, chronic kidney disease, cryptogenic stroke, and a family history of renal failure strengthened the suspicion of Fabry disease. Blood analysis showed borderline low α-galactosidase A activity, leukocyte enzyme activity at the lower limit of normal, and a normal Lyso-Gb3 level. Genetic testing identified the pathogenic p.A143T variant in the *GLA* gene, confirming the diagnosis of Fabry disease. After multidisciplinary evaluation, enzyme replacement therapy was initiated, and family screening identified the same GLA variant in one daughter and one son.

**Conclusion:** This case demonstrates that normal or borderline biochemical test results in female patients do not exclude Fabry disease, and that clinical and imaging red flags should be evaluated together when unexplained left ventricular hypertrophy is accompanied by renal and cerebrovascular findings. While multimodal cardiac imaging plays an important role in strengthening diagnostic suspicion and characterizing myocardial involvement, genetic confirmation is critical for definitive diagnosis and family screening.

**Keywords:** Fabry disease, cryptogenic stroke, left ventricular hypertrophy, cardiac magnetic resonance, multimodal imaging.

## [OP-07]

### Endovascular stent treatment for LVAD outflow graft obstruction: A three-case series

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**Background:** Advanced heart failure carries high morbidity despite optimal therapy; although transplantation remains the gold standard, left ventricular assist devices (LVAD) are increasingly used as bridge-to-transplant, bridge-to-candidacy, or destination therapy due to donor shortages. Outflow graft obstruction, though rare, is a serious mechanical complication causing reduced flow, low-flow alarms, and hemodynamic deterioration through thrombosis, kinking, torsion, or extrinsic compression. Endovascular treatment has increasingly replaced surgical revision, offering high technical success and favorable outcomes. We present three patients treated with endovascular stenting for LVAD outflow graft stenosis.

**Case Reports**

**Case 1:** A 68-year-old man with a HeartWare LVAD (2015, ischemic cardiomyopathy) presented 11 years post-implantation with dyspnea and a low-flow alarm (2760 rpm, 2.1 L/min, 3.6 W). INR/LDH were near-normal; echocardiography excluded thrombus. CT angiography showed stenosis at the aortic anastomosis. A 10x38 mm covered stent (Advanta V12) was implanted via femoral access, improving flow to 4.0 L/min. The patient died one month later of an undetermined cause, unrelated to the procedure.

**Case 2:** A 45-year-old man with a HeartWare LVAD (2018, dilated cardiomyopathy) presented six years later with dyspnea and low flow (5400 rpm, 2.8 L/min), elevated INR (2.7) and LDH (410), initially suggesting pump thrombosis. Poor response to heparin and CT angiography showing ~50% narrowing from neointimal hyperplasia with chronic anastomotic dissection redirected diagnosis to outflow obstruction. Stenting improved flow to 4.8 L/min. The patient remained stable two years, then died from complications following TAVI for aortic regurgitation.

**Case 3:** A 55-year-old man with a HeartWare LVAD (2020, dilated cardiomyopathy) was admitted one year later with altered mental status and a low-flow alarm (2520 rpm, 1.4 L/min). After thrombolysis/heparin failed, CT angiography showed anastomotic stenosis, treated with the same stenting technique, improving flow to 3.4 L/min. The patient remained stable five years before dying of intracranial hemorrhage.

**Discussion:** Outflow graft stenosis and pump thrombosis share overlapping presentations, requiring systematic differential diagnosis. Near-normal LDH without hemolysis favored mechanical obstruction in Cases 1 and 3, while Case 2 initially mimicked thrombosis until failure to respond to heparin prompted imaging. CT angiography was essential for localizing obstruction and guiding intervention, complemented by echocardiography. In all three cases the aortic anastomosis was involved, alone or with neointimal hyperplasia, suggesting chronic mechanical stress and fibrotic healing as key contributors. Given the high risk of redo sternotomy, endovascular stenting offers a less invasive alternative with high success and durable patency, confirmed by sustained flow improvement and absence of restenosis on follow-up.

Obstruction occurred across a wide time range (1-11 years post-implantation), underscoring the need for vigilance regardless of device duration. Limitations include the retrospective, single-center design and small sample size. **Conclusion:** Outflow graft stenosis should be considered in LVAD patients with low pump flow, alongside pump thrombosis. Early CT angiography enables prompt diagnosis, and endovascular stenting is a safe, effective option in suitable candidates, warranting lifelong follow-up.

**Keywords:** Left ventricular assist device, outflow graft obstruction, endovascular stent, pump thrombosis, heart failure, CT angiography, mechanical circulatory support.

## [OP-08]

### Flash pulmonary edema (Pickering syndrome) secondary to bilateral renal artery involvement: A case report

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A 45-year-old female patient presented to emergency department with sudden-onset dyspnea; she was referred to our clinic after findings of volume overload—specifically desaturation, tachypnea, and elevated BNP and troponin levels (142→180)—were detected. Her medical history didn't contain any comorbidities, but she had a 60 pack-year smoking history. Despite the use of multiple high-dose antihypertensive agents, her blood pressure remained uncontrolled (175/96 mmHg), leading to a diagnosis of resistant hypertension. Physical examination revealed an abdominal bruit, rales, and leg pain intermittent claudication. Prominent abdominal systolic murmur was also noticed echocardiography showed an ejection fraction of 60-65% and stage 2 diastolic dysfunction; thoracic CT revealed bilateral pleural effusion and signs of volume overload. Coronary angiography did not reveal critical stenosis; however, contrast induced nephropathy (CIN) developed, manifesting as a rise in creatinine levels from 1.4 to 2.6 mg/dL. The presence abdominal bruit, rapid development of CIN and resistant hypertension prompted a renovascular investigation. Renal and abdominal vascular Doppler ultrasound demonstrated total occlusion of the abdominal aorta distal to the renal arteries. The left renal artery originated from the occluded segment and showed total occlusion of a short proximal segment, while the right renal artery exhibited significant proximal stenosis. Both kidneys supported the presence of post-stenotic flow. Lower extremity arterial Doppler ultrasound showed aortic occlusion at the renal level, with the distal arterial system at the iliac bifurcation level remaining patent via collateral filling. The patient was evaluated by the Rheumatology department for Takayasu arteritis; However, cardiac MRI did not revealed increased wall thickness and luminal narrowing in other large arteries, and PET CT did not show FDG uptake, therefore Takayasu arteritis was not considered. Abdomen BT angiography also confirmed that abdominal aorta was totally occluded distal to the mesenteric artery, the right renal artery was 99% occluded, and the left renal artery could not be visualized. Percutaneous retrograde stents were placed in the bilateral renal arteries via the brachial approach by interventional radiology, and the procedure was completed without complications. Following stent placement, 7 L urine output was done within 24 h, the need for antihypertensive medication decreased, renal function improved (creatinine 1.7→1.0 mg/dL), dyspnea and pleural effusion resolved, and BNP levels dropped from 1646 to 750 pg/mL. The patient has not suffered lung congestion at the 6 month follow-up and only 1 antihypertensive pill has taken. In the joint cardiology-cardiovascular surgery council, no intervention was considered for abdominal aortic occlusion.

**Keywords:** Flash pulmonary edema, hypertension, bilateral renal artery occlusion, Pickering syndrome.

## [OP-09]

### Advanced RV failure in idiopathic pulmonary fibrosis: Is RVAD support the next step?

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**Background:** Pulmonary hypertension (PH) is a major complication of fibrotic interstitial lung disease (ILD) and is associated with poor functional status and increased mortality. Severe pulmonary vascular disease may lead to progressive right ventricular (RV) failure despite treatment of the underlying disease. We present a case of rapidly progressive PH and severe RV dysfunction in a patient with fibrotic ILD demonstrating a usual interstitial pneumonia (UIP) pattern.

**Case Report:** A 67-year-old man with diabetes mellitus, hypertension, fibrotic ILD, PH, and right-sided heart failure was admitted with progressive dyspnea and worsening exercise intolerance. ILD had been diagnosed in 2022 in the setting of suspected antisynthetase syndrome, and he had received azathioprine, cyclophosphamide, mycophenolate mofetil, and corticosteroids. Despite immunosuppressive treatment, respiratory symptoms progressively worsened. Right heart catheterization (RHC) in November 2025 demonstrated PH with pulmonary artery pressure (PAP) 51/22 mmHg (mean, 32 mmHg), pulmonary artery wedge pressure (PAWP) 12 mmHg, cardiac output (CO) 3.49 L/min, cardiac index (CI) 1.86 L/min/m$^2$, and pulmonary vascular resistance (PVR) 5.7 Wood units (WU). Vasoreactivity testing was negative. Macitentan and inhaled iloprost were initiated; macitentan was discontinued because of worsening dyspnea and iloprost after syncope. Rituximab was initiated in May 2026, followed by nintedanib for progressive fibrotic ILD. In August 2026, he was hospitalized with worsening dyspnea, volume overload, and progressive right heart failure. Echocardiography showed preserved LVEF (55%) but severe RV dilatation (53 mm) and dysfunction: TAPSE 8 mm, S′ 7.3 cm/s, RV-FAC 19%, RV free-wall longitudinal strain -8.9%, and RV four-chamber longitudinal strain -6.7%. A D-shaped interventricular septum, right atrial enlargement (4.7 cm), moderate-to-severe tricuspid regurgitation, and pulmonary artery enlargement were present. Estimated systolic PAP was 80 mmHg. Repeat RHC showed marked deterioration: PAP 60/24 mmHg (mean, 42 mmHg), PAWP 5 mmHg, CO 2.50 L/min, CI 1.45 L/min/m$^2$, and PVR 13.5 WU. Mean PAP decreased to 31 mmHg following inhaled iloprost challenge. NT-proBNP was 9897 pg/mL. He was considered INTERMACS profile 4 and required intravenous dobutamine followed by milrinone for 5 days. Inhaled iloprost was subsequently reintroduced six times daily as Group 3 PH therapy. HRCT demonstrated extensive fibrotic UIP, with a main pulmonary artery diameter of 35 mm. RVAD support was initially planned; however, after multidisciplinary discussion with the heart transplantation surgery team, implantation was not pursued.

**Conclusion:** This case demonstrates rapid progression from severe pulmonary vascular disease to advanced RV failure in fibrotic ILD. Over nine months, PVR increased from 5.7 to 13.5 WU and CI declined from 1.86 to 1.45 L/min/m$^2$, accompanied by marked RV dilatation and severe impairment of RV systolic and deformation parameters. The need for sequential inotropic support and consideration of RVAD highlights the therapeutic dilemma of advanced RV failure in severe Group 3 PH. Serial multimodal assessment may be crucial for risk stratification, therapeutic decision-making, and timely evaluation of mechanical circulatory support or transplantation.

**Keywords:** Idiopathic pulmonary fibrosis, hypertension, pulmonary, assisted circulation, hemodynamics, ventricular dysfunction, right.

## [OP-10]

### Successful HeartMate 3 implantation through left thoracotomy as an alternative to redo median sternotomy: A case report

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**Background:** Redo median sternotomy for left ventricular assist device (LVAD) implantation may be hazardous because of mediastinal adhesions, patent coronary bypass grafts, altered anatomy, and previous sternal reconstruction. Thoracotomy-based HeartMate 3 implantation may avoid anterior mediastinal re-entry and reduce the risk of cardiac, vascular, or graft injury. We report exclusive left-thoracotomy HeartMate 3 implantation with outflow graft anastomosis to the descending thoracic aorta in a patient with previous coronary artery bypass grafting (CABG) and rigid sternal reconstruction.

**Case Report:** A 46-year-old man with end-stage ischemic cardiomyopathy had undergone two-vessel CABG complicated by mediastinitis and sternal dehiscence, requiring revision with a rigid plate-and-screw system. Despite optimal medical therapy, he developed progressive heart failure and recurrent hospitalizations. He was in New York Heart Association class IV and INTERMACS profile 4. Echocardiography showed a left ventricular ejection fraction of 15%, moderate mitral regurgitation, mild tricuspid regurgitation, and moderately reduced right ventricular systolic function. Because redo sternotomy was considered high risk, HeartMate 3 implantation through a left anterolateral thoracotomy was planned. After induction of general anesthesia, the patient was intubated with a double-lumen endotracheal tube. Cardiopulmonary bypass was initiated via femoral arterial and venous cannulation. The sewing ring and pump were implanted at the left ventricular apex, and the outflow graft was anastomosed end-to-side to the descending thoracic aorta. The patient was weaned from cardiopulmonary bypass with inotropic support. Postoperatively, inhaled nitric oxide was administered for right ventricular support. After extubation on postoperative day 1, reintubation was required because of respiratory distress and hypoxemia. Following diuretic therapy, he was successfully extubated on postoperative day 2. No re-exploration for bleeding, neurological complication, infection, pump thrombosis, or device dysfunction occurred. He was transferred to the ward on postoperative day 6 and discharged in stable condition on postoperative day 20.

**Discussion:** Previous CABG combined with mediastinitis, sternal dehiscence, and rigid sternal reconstruction made repeat sternotomy particularly hazardous. Left thoracotomy provided direct access to the ventricular apex, while descending thoracic aortic anastomosis avoided dissection of the ascending aorta and anterior mediastinum. Previous reports support the feasibility of thoracotomy-based HeartMate 3 implantation in selected redo patients. However, experience with an exclusive left-thoracotomy approach and descending aortic outflow remains limited. In this patient, the procedure was completed without re-exploration, neurological events, pump thrombosis, infection, or device dysfunction.

**Conclusion:** In this high-risk redo patient, HeartMate 3 implantation was completed exclusively through a left thoracotomy, with the outflow graft anastomosed to the descending thoracic aorta. This approach avoided repeat sternotomy and anterior mediastinal dissection while providing adequate access for pump implantation and outflow graft placement. It may be a practical option in selected patients when reopening the sternum is considered particularly hazardous.

**Keywords:** Heart-assist devices, thoracotomy, aorta, thoracic, reoperation, heart failure.

## [OP-11]

### Simultaneous repair of occult post-infarction left ventricular free-wall rupture and HeartMate 3 implantation

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**Background:** Left ventricular free-wall rupture (LVFWR) is a frequently fatal complication of acute myocardial infarction. Concomitant reconstruction and durable left ventricular assist device implantation are rarely reported.

**Case Report:** A 26-year-old man was transferred 48 hours after anterior myocardial infarction and failed percutaneous treatment of a complete left anterior descending artery occlusion distal to the first diagonal branch. Despite dual inotropes, cardiogenic shock progressed from INTERMACS profile 2 to 1. Echocardiography demonstrated global hypokinesia, left ventricular ejection fraction of 10%, left ventricular end-diastolic diameter of 7.3 cm, moderate right ventricular dysfunction, and a 1.8 cm pericardial effusion compressing the right atrium, without visible myocardial discontinuity. Emergency HeartMate 3 implantation as bridge to transplantation was undertaken. During apical coring, organized thrombus was found sealing an occult 4×5-cm full-thickness anterior LVFWR. After debridement, the defect was reconstructed using a double-layer Dacron patch with circumferential Teflon felt reinforcement, preserving ventricular volume, apical geometry, and inflow orientation. HeartMate 3 implantation was then completed with coaxial inflow positioning, and pump speed was increased from 3000 to 5000 rpm. Cardiopulmonary bypass and cross-clamp times were 130 and 65 minutes. The patient was extubated on postoperative day 1, weaned from inotropes on day 4, and discharged on day 18. At 34 months, he remains alive on support without recurrent rupture, inflow obstruction, device malfunction, pump thrombosis, or major neurologic or hemorrhagic complications.

**Conclusion:** Geometry-preserving patch reconstruction can enable durable HeartMate 3 support despite extensive post-infarction ventricular disruption.

**Keywords:** Free wall rupture, acute MI, ventricular assist device, heart failure.

## [OP-12]

### from residual/recurrent LVOT obstruction to genetic diagnosis: A case of rasopathy-associated hypertrophic cardiomyopathy

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**Background:** Hypertrophic cardiomyopathy (HCM) is most commonly associated with pathogenic variants in sarcomeric genes; however, it may also occur as part of syndromic disorders such as RASopathies. RASopathies, caused by pathogenic variants affecting the RAS/MAPK signaling pathway—particularly Noonan syndrome—represent important phenocopies that may present with an HCM phenotype.

**Case Report:** A 21-year-old woman presented to our clinic with exertional dyspnea. Her medical history revealed that she had undergone septal myectomy at the age of 11 years because of severe LVOT obstruction. Review of the operative records showed that septal muscle resection had been performed through a transaortic approach and that the anterior mitral leaflet had been augmented with autologous pericardium to increase its transverse width. Preoperative echocardiography had demonstrated an interventricular septal thickness of 18 mm, a provoked LVOT gradient of 100 mmHg, systolic anterior motion (SAM) of the mitral valve, and mild mitral regurgitation (MR). Transthoracic echocardiography performed at our clinic

revealed an interventricular septal thickness of 21 mm and a provoked LVOT gradient of 100 mmHg. SAM of the mitral valve and mild MR were present, and the left ventricular ejection fraction was 70%. Laboratory investigations showed normal complete blood count and renal function parameters, whereas the NT-proBNP level was markedly elevated at 11,720 pg/mL (0-125). On physical examination, a grade 3/6 systolic murmur was heard along the left sternal border. In addition, phenotypic features including short stature, hypertelorism, a short and broad neck, and ptosis were noted. Given the possibility of syndromic HCM, genetic testing was performed. Genetic analysis identified a heterozygous pathogenic variant in the *RAF1* gene, establishing the diagnosis of Noonan syndrome.

**Discussion:** Noonan syndrome is a RASopathy caused by pathogenic variants that result in dysregulation of the RAS/MAPK signaling pathway. Abnormal activation of this pathway promotes cardiomyocyte growth and enhances the hypertrophic response, thereby contributing to the development of the HCM phenotype. Compared with sarcomeric HCM, RASopathy-associated HCM typically manifests at an earlier age, is more frequently accompanied by ventricular outflow tract obstruction, and is associated with higher rates of hospitalization and intervention. Similarly, severe recurrent LVOT obstruction years after childhood septal myectomy has recently been reported in an adult patient with RAF1-associated Noonan syndrome. In that patient, cardiac myosin inhibition, rather than repeat septal reduction therapy, resulted in substantial reductions in the LVOT gradient and symptom burden. Another case report involving a patient with RASopathy-associated HCM demonstrated that treatment with the MEK inhibitor trametinib reduced LVOT obstruction and NT-proBNP levels.

**Conclusion:** In patients who underwent septal myectomy during childhood and present with residual or recurrent LVOT obstruction in adulthood, the original diagnosis of HCM should be reconsidered. Syndromic cardiomyopathies should be included in the differential diagnosis, and appropriate patients should be referred for genetic evaluation. This case illustrates that comprehensive echocardiographic and phenotypic assessment may guide clinicians toward the correct etiological diagnosis, even many years after the initial presentation.

**Keywords:** Noonan syndrome, hypertrophic cardiomyopathy, genetic testing, septal myectomy.

## [OP-13]

### Clinical recovery after classical quadruple medical therapy in advanced heart failure

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**Background:** Advanced heart failure is a condition that leads to serious morbidity and mortality when it is not treated appropriately and in a timely manner. This case report describes a patient who remained untreated for a long time after diagnosis, presented with severe valvular regurgitation and a low ejection fraction, and showed marked clinical improvement within a short time after standard quadruple therapy.

**Case Report:** A 56-year-old man was diagnosed with heart failure in 2021 but had not received appropriate treatment until now. Cardiac MRI showed an interventricular septal thickness of 1.3 cm, diffuse mid-myocardial enhancement of the left ventricle, and enlarged left ventricular dimensions. Transthoracic echocardiography revealed enlarged left heart chambers, severe mitral regurgitation, and severe tricuspid regurgitation, and the ejection fraction was assessed as 25%. A PYP scintigraphy requested for etiological differentiation because of the septal thickness was Grade 0, and no finding in favor of cardiac amyloidosis was identified. A genetic test targeting the etiology was requested, and the result is awaited. The NT-proBNP requested at the initial presentation could not be measured for technical reasons, and no previous BNP value was available for the patient. Marked clinical recovery was observed within one month in the patient after standard quadruple heart failure therapy was started. The follow-up NT-proBNP was 1440 pg/mL, and the strain values showed a certain degree of improvement compared with baseline. The severe valvular regurgitations did not change over the one-month period.

**Discussion and Conclusion:** This case shows that even in patients with advanced heart failure in whom treatment was started late, standard quadruple therapy can achieve significant clinical improvement in a short time. In the presence of a thickened interventricular septum, excluding infiltrative causes such as amyloidosis with PYP scintigraphy is important for the differential diagnosis. The absence of change in valvular regurgitation over a short period suggests that the improvement occurred mainly through myocardial function, and a certain degree of recovery was observed in LV strain on transthoracic echocardiography. The case once again emphasizes the importance of early and maximal therapy in advanced heart failure.

**Keywords:** Quadruple therapy, strain echocardiography, advanced heart failure.

## [OP-14]

### Management of lead-related superior vena cava syndrome after heart transplantation: A case report

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**Background:** Superior vena cava syndrome (SVCS) results from obstruction of venous return through the superior vena cava (SVC). Although malignancies account for 60-70% of cases, benign causes, particularly cardiac implantable electronic device leads, are increasingly recognized. We report lead-related SVCS becoming overt after heart transplantation in a young patient with hypertrophic obstructive cardiomyopathy and prior class I indications for ICD implantation.

**Case Report:** A 31-year-old man with an ICD implanted eight years earlier developed bilateral innominate vein stenosis after two years; because transvenous access was unsuitable, epicardial leads were implanted and the malfunctioning transvenous leads were abandoned. He remained minimally symptomatic for six years. During subsequent orthotopic heart transplantation, most device material was removed, but a chronically adherent lead fragment within the SVC/left innominate region was retained because of the risk of vascular injury. On postoperative day 3, severe head, neck, and bilateral upper-extremity edema developed. Venography demonstrated occlusion of both innominate veins and the cranial SVC, extensive collateralization, and a patent SVC-right atrial anastomosis. Recanalization attempts failed; balloon angioplasty of a collateral pathway produced no significant clinical improvement. Rivaroxaban 20 mg/day was then initiated but was ineffective after two weeks. Therapy was changed to warfarin (target INR 2.0-3.0) with low-molecular-weight heparin bridging. Approximately one week after therapeutic INR was achieved, edema markedly regressed. No recurrence occurred during six months of follow-up.

**Discussion:** Chronic transvenous leads can cause endothelial injury, thrombosis, and fibrotic venous remodeling, while collateral development often prevents symptoms. In this case, transplantation likely disrupted this compensated state through mediastinal dissection and SVC manipulation. Retained lead fragments after transplantation are clinically relevant because of thrombotic and access-related complications and possible adverse outcomes. Although post-transplant SVC obstruction may be treated by angioplasty/stenting, lead extraction with venoplasty ± stenting is commonly used for lead-related SVCS, with anticoagulation remaining important when intervention is unsuitable. DOAC-calcineurin inhibitor interactions and limited transplant-specific evidence should be considered, while corticosteroids may alter warfarin response. Successful DOAC-only treatment has been reported

in thrombosis-dominant cases, but responses may differ in chronic fibrotic occlusion.

**Conclusion:** Heart transplantation may precipitate overt SVCS in previously compensated lead-related central venous occlusion. Preoperative venous mapping, careful management of retained leads, postoperative surveillance, and individualized multidisciplinary treatment are essential.

**Keywords:** Superior vena cava syndrome, heart transplantation, implantable defibrillators, venous thrombosis, anticoagulants.

## [OP-15]

### Massive posterior LV free wall hypertrophy presenting with sustained ventricular tachycardia

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**Patient Presentation:** A 37-year-old man was referred after incidental detection of heterogeneous left ventricular metabolic activity on positron emission tomography-computed tomography performed for oncological surveillance. He had a history of surgically treated neuroendocrine carcinoma in remission. He reported progressive exertional dyspnea, orthopnea, palpitations and reduced exercise tolerance. There was no history of syncope or previously diagnosed cardiomyopathy, and family history was negative for hypertrophic cardiomyopathy or sudden cardiac death. On examination, he was tachycardic and orthopneic, with bibasal crackles and an apical systolic murmur.

**Initial Assessment:** Electrocardiography demonstrated sustained monomorphic ventricular tachycardia at 134 beats/min. N-terminal pro-B-type natriuretic peptide was elevated. Transthoracic echocardiography revealed marked asymmetric left ventricular hypertrophy predominantly involving the posterior free wall, with only mild septal thickening. There was no systolic anterior motion or left ventricular outflow tract obstruction. Moderate mitral regurgitation and grade II diastolic dysfunction were present.

**Diagnosis and Treatment:** Cardiac magnetic resonance confirmed massive asymmetric hypertrophy of the posterior left ventricular free wall, reaching 66 mm, with a left ventricular ejection fraction of 52%. Late gadolinium enhancement showed limited focal mid-myocardial fibrosis involving approximately 3% of left ventricular mass. Increased native T1 and T2 values with mild extracellular volume expansion suggested concomitant myocardial inflammation. Genetic testing identified an NF1 variant of uncertain significance. The final diagnosis was non-obstructive hypertrophic cardiomyopathy with massive posterior free wall involvement complicated by sustained ventricular tachycardia. Sinus rhythm was restored with intravenous amiodarone, and an implantable cardioverter-defibrillator was implanted for secondary prevention according to ESC guidelines. Guideline-directed medical therapy was initiated.

**Follow-up:** The patient was readmitted with recurrent ventricular tachycardia and electrical storm despite medical therapy. Device interrogation confirmed multiple appropriate implantable cardioverter-defibrillator therapies. Catheter ablation was attempted, but ventricular tachycardia remained refractory. Hemodynamic deterioration required intubation and veno-arterial extracorporeal membrane oxygenation. He was transferred to an advanced heart failure center for urgent heart transplantation evaluation.

**Conclusion:** This case highlights the malignant arrhythmic potential of rare non-septal hypertrophic cardiomyopathy phenotypes. Cardiac magnetic resonance was essential for characterizing massive posterior free wall hypertrophy and associated tissue abnormalities. Severe ventricular arrhythmias may occur despite limited fibrosis, potentially exacerbated by concomitant myocardial inflammation. Early risk assessment, secondary-prevention implantable cardioverter-defibrillator implantation and timely referral to advanced heart failure and transplantation centres are crucial in rapidly progressive cases.

**Keywords:** Hypertrophic cardiomyopathy, ventricular tachycardia, cardiac magnetic resonance.

## [OP-16]

### Multimorbidity burden across the LVEF spectrum in Cameroonian HF patients

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**Background:** Multimorbidity is common in heart failure (HF), complicates treatment, and is associated with worse clinical outcomes. There is a paucity of data on the burden of multimorbidity in patients with HF across the left ventricular ejection fraction (LVEF) spectrum in Cameroon. To determine the prevalence of multimorbidity, the difference in prescription of guideline-directed medical therapy (GDMT) between multimorbidity and no multimorbidity, and its effect on in-hospital mortality and length of hospital stay in patients hospitalized with HF across the LVEF spectrum at two major hospitals in Cameroon.

**Methods:** A five-year (1st January 2020-1st January 2025) hospital-based retrospective cross-sectional study was conducted at the Buea Regional Hospital and Douala General Hospital. Medical records of eligible adult patients admitted with a primary diagnosis of HF were reviewed. Patients were classified by HF phenotype according to the 2021 European Society of Cardiology (ESC) Guidelines. Multimorbidity was quantified using a simple comorbidity count index. Categorical variables were compared using the chi-square test, and continuous variables with ANOVA. Binary logistic regression was used to identify independent predictors of mortality and GDMT prescription. Statistical significance was set at $p<0.05$.

**Results:** A total of 365 patients were included (mean age 61.6±16.1 years; 58.1% female). The dominant HF phenotype was HFrEF (72.9%), followed by HFpEF (17.0%) and HFmrEF (10.1%). The overall prevalence of multimorbidity (≥2 comorbidities) was 64.9%. Highest in HFmrEF (81.1%). The most prevalent comorbidities were hypertension (66.8%), anemia (36.2%), chronic kidney disease (23.3%), and diabetes mellitus (20.0%). Multimorbid patients were more likely to receive SGLT2 inhibitors (14.8% *vs.* 6.3%; p=0.016) but less likely to receive mineralocorticoid receptor antagonists (48.1% *vs.* 62.5%; p=0.009). Optimal four-pillar GDMT was achieved in only 5.2% of patients. In-hospital mortality was 11.0% (n=38). Although multimorbidity was associated with higher rates of discharge against medical advice (7.2% *vs.* 2.3%; p=0.154), prolonged hospital stay (>7 days) did not differ significantly between groups (60.9% *vs.* 57.4%; p=0.154). Each additional comorbidity independently predicted mortality (OR=1.26; 95% CI: 1.01-1.60; p=0.040). Atrial fibrillation (OR=2.51; p=0.014) and chronic kidney disease (OR=2.48; p=0.016) were the strongest independent predictors of death, whereas hypertension was paradoxically protective (OR=0.46; p=0.030).

**Conclusion:** Multimorbidity is highly prevalent among HF patients in Cameroon, affects nearly two-thirds of those hospitalized, and independently predicts in-hospital mortality. Integrated, patient-centred care models addressing the full spectrum of comorbid conditions are urgently needed to improve outcomes in this high-risk population.

**Keywords:** Heart failure, multimorbidity, LVEF spectrum, guideline-directed medical therapy, in-hospital mortality, Cameroon.

## [OP-17]

### Prognostic impact of valvular regurgitation in chronic heart failure: Crude associations explained by disease severity

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**Background:** Moderate-to-severe mitral (MR) and tricuspid regurgitation (TR) are common in chronic heart failure (HF) and are frequently described as markers of adverse outcome. Whether significant regurgitation carries prognostic information beyond conventional indices of HF severity, and whether the number of affected valves adds incremental risk, remains uncertain. We examined the association between regurgitation burden and hard clinical outcomes before and after adjustment for established prognostic variables.

**Methods:** Consecutive patients with chronic HF undergoing transthoracic echocardiography were analyzed retrospectively. MR, TR and aortic regurgitation (AR) were graded by an integrated multiparametric approach, and a lesion was considered significant at grade ≥2 (moderate or greater). Regurgitation burden was defined as the number of valves with ≥2 grade regurgitation (0, 1, or ≥2). The primary endpoint was a composite of all-cause mortality, HF hospitalization, and LVAD implantation or heart transplantation. Each lesion was first examined separately, then all three lesions were entered simultaneously (mutually adjusted model), and finally a fully adjusted logistic model was fitted including age, left ventricular ejection fraction (LVEF), baseline NYHA class, log-transformed BNP and serum creatinine. Burden was additionally modelled as a continuous variable with a test for trend. A two-sided $p<0.05$ was considered significant.

**Results:** Significant regurgitation was frequent: ≥2 MR in 146/560 patients (26.1%), ≥2 TR in 102/561 (18.2%) and ≥2 AR in 25/574 (4.4%). In unadjusted analysis, both ≥2 MR (OR 2.72, 95% CI 1.78-4.16, $p<0.001$) and ≥2 TR (OR 2.98, 95% CI 1.87-4.75, $p<0.001$) were associated with the composite endpoint, whereas ≥2 AR was not (OR 0.88, 95% CI 0.33-2.41, $p=0.810$). MR and TR remained associated with each other's effect held constant (OR 1.91 and 2.41, respectively). Event rates rose stepwise with regurgitation burden—14.6% with no significant lesion, 30.0% with one affected valve and 40.8% with ≥2 affected valves (OR 2.51 and 4.05 versus none; p for trend <0.001)—and the combination of MR and TR carried the highest event rate (46.7%). After adjustment for age, LVEF, NYHA class, ln(BNP) and creatinine, none of the individual lesions (≥2 MR: OR 1.28, $p=0.451$; ≥2 TR: OR 1.24, $p=0.546$; ≥2 AR: OR 0.69, $p=0.551$) nor regurgitation burden as a continuous variable (OR 1.18 per additional valve, $p=0.355$) remained independently associated with outcome. Lower LVEF (OR 0.97 per 1% increase, $p=0.044$), higher NYHA class (OR 2.12 per class, $p<0.001$) and higher ln(BNP) (OR 1.29, $p=0.014$) were the only independent predictors.

**Conclusion:** In this HF cohort, moderate-to-severe MR and TR and a rising burden of significant regurgitation identified patients at markedly higher risk of death, HF hospitalization or LVAD/transplantation, with a clear dose-response pattern. These crude associations were, however, entirely accounted for by ventricular function, functional class and natriuretic peptide level. Significant atrioventricular regurgitation in chronic HF therefore appears to behave as a marker of advanced disease—reflecting remodelling, elevated filling pressures and impaired functional capacity—rather than as an independent driver of outcome. This distinction is relevant when regurgitation severity alone is used to select patients for valve-directed therapy.

**Keywords:** Heart failure, mitral regurgitation, tricuspid regurgitation, valvular regurgitation burden, prognosis.

## [OP-18]

### Combined sST2 and NT-proBNP assessment for left ventricular dysfunction in heart failure

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**Background:** Heart failure (HF) is a complex clinical syndrome characterized by multiple pathophysiological mechanisms, including myocardial wall stress, inflammation, fibrosis, and adverse ventricular remodeling. N-terminal pro-B-type natriuretic peptide (NT-proBNP) is the current reference biomarker reflecting myocardial stretch, whereas soluble suppression of tumorigenicity-2 (sST2) reflects myocardial fibrosis and inflammatory activation. Since these biomarkers represent distinct biological pathways, their combined evaluation may provide a more comprehensive assessment of cardiac dysfunction than either biomarker alone. We aimed to investigate the relationship of baseline sST2 and NT-proBNP with left ventricular ejection fraction (LVEF) in patients with heart failure.

**Methods:** This single-center cross-sectional study included 100 consecutive patients with a diagnosis of heart failure. Demographic, clinical, laboratory, and echocardiographic data were collected at baseline. Serum sST2 and NT-proBNP concentrations were measured on admission, and LVEF was determined using transthoracic echocardiography. Correlations between biomarkers and LVEF were evaluated using Spearman correlation analysis. Patients were additionally categorized according to heart failure phenotype based on LVEF: heart failure with reduced ejection fraction (HFrEF), mildly reduced ejection fraction (HFmrEF), and preserved ejection fraction (HFpEF). Differences in biomarker concentrations among groups were assessed using non-parametric statistical tests. Multivariable linear regression analysis was performed to determine whether sST2 and NT-proBNP were independently associated with LVEF after adjustment for age and other clinically relevant variables.

**Results:** Baseline sST2 demonstrated a significant inverse correlation with LVEF, indicating that increasing sST2 concentrations were associated with worsening left ventricular systolic function. NT-proBNP also showed an inverse relationship with LVEF, consistent with its established role as a marker of myocardial wall stress. Patients with reduced LVEF exhibited higher circulating concentrations of both biomarkers than those with preserved ventricular function. In multivariable analysis, the combined assessment of sST2 and NT-proBNP improved the characterization of left ventricular systolic dysfunction compared with evaluation based on either biomarker alone. The complementary biological information provided by fibrosis-related and hemodynamic biomarkers supported a more comprehensive evaluation of disease severity. These findings are consistent with previous studies demonstrating that combining sST2 with NT-proBNP enhances assessment of HF severity and prognosis.

**Conclusion:** The combined measurement of sST2 and NT-proBNP may provide complementary information regarding left ventricular systolic dysfunction in patients with heart failure. Integrating biomarkers reflecting myocardial fibrosis and wall stress has the potential to improve phenotypic characterization and clinical risk assessment beyond the use of a single biomarker. Larger prospective studies are warranted to validate these findings and to establish the incremental value of multimarker strategies in routine clinical practice

**Keywords:** sST2, NT-proBNP, LVEF.

## [OP-19]

### Patterns and etiology of hospital readmission after continuous-flow left ventricular assist device implantation

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**Background:** Hospital readmission is common after left ventricular assist device (LVAD) implantation and adversely affects quality of life, cost, and survival. Contemporary data describing readmission patterns, etiology, and patients' characteristics are needed. We characterized the burden, timing, etiology, and determinants of readmission in an INTERMACS-based cohort.

**Methods:** We conducted a retrospective cohort study of 40 adults who had primary continuous-flow LVAD implantation from 2021 to 2025. The cumulative incidence of a first readmission was estimated using Kaplan-Meier; the recurrent readmission rate was modeled with an Andersen-Gill counting-process model with patient-clustered robust standard errors, adjusted for body-mass index (BMI) and right-ventricular (RV) function, with Prentice-Williams-Peterson, shared-frailty, and negative binomial sensitivity analyses.

**Results:** Over 62.2 patient-years (median follow-up 15.9 months), 21 patients (52%) were readmitted at least once, and 16 (40%) had repeated admissions, totaling 46 readmissions (0.74 per patient-year). The cumulative incidence of a first readmission was 28%, 54%, and 65% at 6, 12, and 18 months. Major infection was the leading cause (33/46, 72%), and most readmissions were managed conservatively (70%). Readmitted patients had higher BMI (p=0.030) and body surface area (p=0.002). Neither BMI (hazard ratio 1.01, 95% CI 0.96-1.05) nor RV function (1.08, 0.87-1.34) independently predicted the readmission rate.

**Conclusion:** Readmissions after LVAD were frequent, recurrent, and infection-driven, and concentrated in a subset of patients. They were more common in patients with higher BMI. Findings are hypothesis-generating and support intensified infection-prevention and transitionalcare strategies.

**Keywords:** Left ventricular assist device, hospital readmission, recurrent events, driveline/device-related infection.

## [OP-21]

### Early postoperative exercise therapy in HFrEF cardiac surgery

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**Background:** Supervised early postoperative exercise therapy is recommended after cardiac surgery, but the foundational trials systematically excluded patients with heart failure and reduced ejection fraction (HFrEF). We evaluated whether this therapy can be delivered safely and effectively to HFrEF patients undergoing open cardiac surgery.

**Methods:** Single-center retrospective pre-post cohort study (January 2024 to April 2026). Of 1,058 consecutive open cardiac surgery patients, 62 had HFrEF (LVEF <40%) and formed the primary cohort: 28 operated before and 34 after the introduction of a supervised early exercise therapy program in February 2025. The program comprised preoperative education, early-extubation mobilization, twice-daily respiratory and limb exercises in the intensive care unit, and ward-based progressive ambulation. Patients with preserved LVEF (≥50%, n=821) treated under the same protocol served as an internal benchmark; the remaining 175 patients had mid-range LVEF (40-49%) and were not included in either stratum. The primary safety endpoint was in-hospital mortality; the primary effectiveness endpoint was reintubation. Prolonged intensive care unit stay was defined as a postoperative intensive care duration exceeding 72 hours, in keeping with a recent isolated coronary artery bypass grafting cohort. Multivariable logistic regression adjusted for age, sex, ASA grade, diabetes mellitus, hypertension, and chronic kidney disease.

**Results:** Isolated coronary artery bypass grafting accounted for 90.3% (56/62) of the HFrEF cohort. In-hospital mortality was 17.9% before versus 20.6% after program introduction (p=1.00). Reintubation was 10.7% versus 5.9% (p=0.65), a difference of one patient (3 versus 2 events) and therefore exploratory. Prolonged intensive care unit stay was not reduced (adjusted OR 1.30, 95% CI 0.33 to 5.14, p=0.71). In the preserved-ejection-fraction benchmark the same protocol reduced prolonged intensive care unit stay (adjusted OR 0.60, 95% CI 0.41 to 0.88, p=0.008).

**Discussion:** Introducing supervised early exercise therapy produced no detectable mortality or reintubation signal in HFrEF patients, while the concurrent preserved-ejection-fraction benchmark showed that the protocol itself was effective within the same institutional system. This contrast argues against implementation failure and suggests that resource-utilization gains observed in preserved-ejection-fraction populations should not be extrapolated to HFrEF. The single-center, retrospective pre-post design and the small number of events limit inference, and adherence was not formally audited.

**Conclusion:** No safety signal was detected when supervised early postoperative exercise therapy was extended to HFrEF cardiac surgery patients, although the small number of events precludes a formal safety conclusion. The intensive care unit stay benefit seen with preserved ejection fraction should not be assumed in this population. Prospective HFrEF-specific protocols with individualized dosing are needed.

**Keywords:** Heart failure, stroke volume, cardiac surgical procedures, coronary artery bypass, exercise therapy, early ambulation.

## [OP-22]

### Acetazolamide in company of other treatment and their effect on serum chloride and NT-proBNP levels: Observational research

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In recent literature, chloride is emerging as an important electrolyte to pay attention to in heart failure. Beyond its passive role in maintaining electroneutrality and osmotic balance, chloride actively contributes to renal tubular transport via the $Na^+$-$Cl^-$ cotransporter (NCC) and $Na^+$-$K^+$-$2Cl^-$ cotransporter (NKCC), regulates acid-base homeostasis through bicarbonate exchange, and modulates neurohormonal activity by influencing renin release at the macula densa. Multiple studies have shown an association between hypochloremia and an increased risk of HF episodes, as well as cardiovascular and all-cause mortality, in both acute and chronic HF settings. These relationships remain robust even after comprehensive adjustment for confounding factors, including hyponatremia, renal dysfunction, and other established prognostic markers. But there is limited data on whether treating hypochloraemia has positive effects on these endpoints or whether there is a treatment goal for chloride levels similar to potassium or sodium levels. Coincidentally, acetazolamide is emerging as a new cornerstone of decongestion therapy in acute congestive heart failure, which, by its mechanistic nature, increases serum chloride levels. In contrast, many other decongestive therapies in heart failure decrease serum chloride. Acetazolamide in the company of treatments and their effect on serum chloride and NT-proBNP levels: An observational research (ACE- CLOR) is an observational study studying the relation of chloride and nt-proBNP course during hospitalization for acute decompensated heart failure in patients receiving oral acetazolamide. Preliminary results of current 90 included patients show no statistical relation of chloride to nt-proBNP but patients who had corrected hypochloremia had greater nt-proBNP reduction

percentage that patients who remained hypochloremic, greater patient population might show promising results.

**Keywords:** Chloride, heart failure, acute decompensated heart failure, acetazolamide, diuretic.

## [OP-23]

### Impact of diabetes duration and glycemic control on left ventricular ejection fraction in patients with heart failure

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**Background:** Although type 2 diabetes mellitus (T2DM) is an important risk factor for the development and progression of heart failure (HF), the independent effects of diabetes duration and glycemic control on left ventricular systolic function have not been sufficiently clarified. This study aimed to evaluate the relationship of diabetes duration and glycemic control with left ventricular ejection fraction (EF) in patients with coexisting T2DM and HF, and to identify other clinical determinants associated with low EF.

**Methods:** This single-center, retrospective study included a total of 1,265 patients who were hospitalized for decompensated HF between January 2010 and January 2022, were discharged after treatment, and had a diagnosis of T2DM before the index admission. Patients were divided into two groups according to left ventricular EF: HFrEF (EF ≤40%) and HFmrEF+HFpEF (EF >40%). The groups were compared with respect to demographic and clinical characteristics, comorbidities, laboratory parameters, medical therapies, HbA1c levels, and diabetes duration. Univariable and multivariable logistic regression analyses were performed to identify independent variables associated with low EF. The performance of diabetes duration in discriminating low EF was assessed by receiver operating characteristic (ROC) analysis.

**Results:** Of the 1,265 patients included, 697 had HFrEF and 568 had HFmrEF or HFpEF. Diabetes duration was significantly longer in the HFrEF group than in patients with EF >40% (13 *vs.* 7 years; p<0.001). Similarly, HbA1c levels were significantly higher in the HFrEF group (8.4±1.6% *vs.* 7.7±1.5%; p<0.001). However, in multivariable logistic regression analysis, diabetes duration (OR: 2.23; p<0.001) and the presence of hypertension (OR: 6.62; p<0.001) were identified as independent determinants of low EF, whereas the use of oral antidiabetic therapy was associated with a lower likelihood of low EF (OR: 0.74; p=0.042). Although HbA1c level was significant in univariable analysis, it lost its status as an independent determinant in multivariable analysis (OR: 1.05; p=0.269). In ROC analysis, the area under the curve of diabetes duration for discriminating low EF was 0.780 (p<0.001). A diabetes duration cut-off of 8.5 years showed 75% sensitivity and 70% specificity for identifying low EF.

**Conclusion:** In patients with coexisting T2DM and HF, longer diabetes duration was associated with low left ventricular EF, independently of the current level of glycemic control and the other clinical risk factors evaluated. In contrast, although HbA1c levels were higher in the HFrEF group, HbA1c was not an independent determinant of low EF. These findings indicate that cardiac risk assessment in diabetic patients should not rely solely on current glycemic control indicators, and underscore the importance of considering diabetes duration, as a marker of cumulative metabolic exposure, in cardiovascular evaluation. Early and comprehensive cardiovascular evaluation in patients with long-standing T2DM may be clinically important for the timely detection of left ventricular dysfunction.

**Keywords:** Heart failure, type 2 diabetes mellitus, diabetes duration, glycemic control, ejection fraction.

## [OP-24]

### Hemocompatibility outcomes after transition to apixaban in HeartMate 3 recipients

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**Background:** Warfarin remains the standard anticoagulation therapy for patients supported with the HeartMate 3 (HM3) left ventricular assist device. However, maintaining a stable INR and managing bleeding complications can be difficult during long-term treatment. We evaluated clinical outcomes and hemolysis markers after switching from warfarin to apixaban in HM3 recipients with poor anticoagulation control and/or bleeding complications.

**Methods:** We retrospectively reviewed eight HM3 recipients who were switched from warfarin to apixaban at our center. Seven patients received apixaban 5 mg twice daily and one received 2.5 mg twice daily. All patients continued aspirin therapy. Time in therapeutic range (TTR) during the six months before the switch, warfarin-related complications, and the reason for switching to apixaban were recorded. Major bleeding, ischemic stroke or transient ischemic attack, systemic embolism, and pump thrombosis were assessed during follow-up. Hemoglobin and lactate dehydrogenase (LDH) levels were recorded at baseline and at 3, 6, and 12 months. Paired laboratory values were compared using the Wilcoxon signed-rank test.

**Results:** The mean age was 63.1±7.5 years, and six patients (75%) were male. Median TTR during the six months before switching to apixaban was 31% (range, 15-35%). While receiving warfarin, six patients had major bleeding and two had non-major bleeding. Seven patients were switched because of bleeding complications, while one was switched because of difficulties with warfarin monitoring and adherence. Median follow-up after switching to apixaban was 14 months (IQR, 13-16 months). No major bleeding, ischemic stroke or transient ischemic attack, systemic embolism, or pump thrombosis was observed during follow-up. Twelve-month LDH measurements were available for all eight patients. Mean LDH decreased from 206.4±40.8 U/L at baseline to 177.4±34.9 U/L at 12 months, and LDH decreased in every patient. The change was significant on paired analysis (p=0.038). Among the six patients with 12-month hemoglobin measurements, mean hemoglobin increased from 10.15±2.31 g/dL at baseline to 11.62±2.78 g/dL at 12 months, although the difference was not statistically significant (p=0.156).

**Discussion and Conclusion:** Patients switched to apixaban in this series had poor anticoagulation control and frequent bleeding complications while receiving warfarin. During a median follow-up of 14 months, no major bleeding, ischemic stroke or transient ischemic attack, systemic embolism, or pump thrombosis was observed. LDH decreased in all eight patients at 12 months, with no laboratory evidence of increased hemolysis after the switch to apixaban. Although these results are encouraging, the small number of patients and the lack of a warfarin control group do not allow conclusions regarding the comparative safety or efficacy of apixaban. Further experience in larger HM3 populations is needed.

**Keywords:** Heart-assist devices, apixaban, warfarin, hemolysis.

## [OP-25]

### Blood pressure control, clinical characteristics, and guideline-directed medical therapy in patients with heart failure

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**Background:** Optimal blood pressure (BP) management is an important component of heart failure (HF) care. However, BP control may vary according to HF phenotype, comorbidities, and treatment patterns. This study aimed to determine BP control rates and compare clinical characteristics and guideline-directed medical therapy (GDMT) patterns between HF patients with controlled and uncontrolled BP.

**Methods:** A total of 2,272 patients with HF were included in the registry. BP control status was available for 2,203 patients. Patients were categorized as having controlled or uncontrolled BP according to the predefined study variable. Uncontrolled BP was defined as an office systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg. Demographic and clinical characteristics, HF phenotype, functional status, comorbidities, cardiovascular interventions, and HF medications were compared between the two groups. Medication data were available for 2,189 patients.

**Results:** Among 2,203 patients with available BP data, 1,858 (84.3%) had controlled BP and 345 (15.7%) had uncontrolled BP. HF phenotype differed significantly between groups. HFrEF was less frequent among patients with uncontrolled BP (55.7% *vs.* 66.6%), whereas HFmrEF (13.9% *vs.* 9.3%) and HFpEF (30.4% *vs.* 24.2%) were more frequent. Uncontrolled BP was also associated with differences in cardiovascular comorbidity profile. Coronary artery disease (53.3% *vs.* 59.0%, p=0.048) and peripheral artery disease (2.3% *vs.* 4.7%, p=0.043) were less frequent among patients with uncontrolled BP. Significant differences were observed in HF pharmacotherapy. Compared with patients with controlled BP, those with uncontrolled BP had lower use of beta-blockers (85.7% *vs.* 91.2%, p=0.001), sacubitril/valsartan (7.0% *vs.* 11.9%, p=0.008), mineralocorticoid receptor antagonists (48.7% *vs.* 63.7%, p<0.001), and SGLT2 inhibitors (37.0% *vs.* 49.9%, p<0.001). Four-class GDMT use was also less frequent in patients with uncontrolled BP (20.1% *vs.* 32.1%; p<0.001).

**Conclusion:** BP was controlled in 84.3% of patients with HF, while 15.7% had uncontrolled BP. Patients with uncontrolled BP had a higher proportion of HFpEF and HFmrEF and lower use of several components of GDMT. These findings highlight differences in HF phenotype and GDMT use according to BP control status and underscore the need for individualized BP and HF treatment strategies.

**Keywords:** Heart failure, blood pressure control, guideline-directed medical therapy, heart failure phenotypes, hypertension.

## [OP-26]

### Clinical and phenotypic characteristics according to NOAC selection in patients followed at heart failure centers

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**Background:** Non-vitamin K antagonist oral anticoagulants (NOACs) are widely used in patients with heart failure (HF) requiring oral anticoagulation. In routine clinical practice, the selection of a specific NOAC may be influenced by patient phenotype, comorbidities, and renal function. This study aimed to compare clinical and phenotypic characteristics according to the prescribed NOAC in patients followed at heart failure centers.

**Methods:** This cross-sectional analysis included 630 HF patients receiving a NOAC. Patients were categorized according to the prescribed agent as apixaban, dabigatran, edoxaban, or rivaroxaban. Demographic characteristics, HF phenotype and duration, NYHA functional class, cardiovascular risk factors, renal function, comorbidities, previous cardiovascular interventions, and concomitant HF therapies were compared across NOAC groups using the chi-square test.

**Results:** Among 630 patients receiving a NOAC, 260 (41.3%) were treated with apixaban, 261 (41.4%) with rivaroxaban, 100 (15.9%) with edoxaban, and 9 (1.4%) with dabigatran. The distribution of HF phenotypes was similar across NOAC groups (p=0.523); HFrEF was present in 49.6% of apixaban-, 66.7% of dabigatran-, 52.0% of edoxaban-, and 53.6% of rivaroxaban-treated patients. NYHA functional class (p=0.428) and HF duration (p=0.506) also did not differ significantly between treatment groups. Likewise, no significant differences were observed for sex (p=0.088), hypertension (p=0.253), diabetes mellitus (p=0.205), coronary artery disease (p=0.524), atrial fibrillation/flutter (p=0.989), anemia (p=0.640), valvular heart disease (p=0.253), or malignancy (p=0.370). In contrast, renal function category differed significantly according to the selected NOAC (p=0.048), suggesting that renal phenotype may contribute to anticoagulant selection. Smoking status also differed significantly among NOAC groups (p=0.006); current or former smoking was more frequent among patients receiving rivaroxaban and edoxaban than

among those receiving apixaban. No significant differences were observed in overall guideline-directed HF therapy burden across NOAC groups (p=0.609).

**Conclusion:** In patients followed at heart failure centers, NOAC selection was not significantly associated with HF phenotype, functional status, or most major cardiovascular comorbidities. Renal function category differed significantly across NOAC groups, although this finding requires confirmation in adjusted analyses. The observed association with smoking status requires further evaluation and may reflect differences in the underlying clinical profile rather than a direct determinant of drug selection.

**Keywords:** Heart failure, NOAC, apixaban, rivaroxaban, edoxaban, dabigatran, renal function, anticoagulation.

## [OP-27]

### Clinical characteristics and comorbidity burden of heart failure patients with sleep apnea

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**Background:** Sleep apnea is frequently associated with cardiovascular disease and may contribute to symptom burden and comorbidity in patients with heart failure (HF). We aimed to compare clinical characteristics, HF phenotype, functional status, and comorbidity burden between HF patients with and without documented sleep apnea.

**Methods:** Among 2,259 patients in whom sleep apnea status was recorded, 64 had documented sleep apnea, 1,745 had no documented sleep apnea, and sleep apnea status was unknown in 450 patients. Patients with unknown sleep apnea status were excluded from the comparative analysis. Clinical characteristics and comorbidities were compared between patients with and without sleep apnea.

**Results:** A total of 1,809 patients with known sleep apnea status were included in the comparative analysis. The proportion of male patients was numerically higher in the sleep apnea group (71.9% *vs.* 61.8%, p=0.102). The distribution of HF phenotypes did not differ significantly between groups (p=0.108), although HF with preserved ejection fraction was numerically more frequent in patients with sleep apnea (39.1% *vs.* 27.3%), whereas HF with reduced ejection fraction was less frequent (50.0% *vs.* 61.8%). NYHA functional class differed significantly according to sleep apnea status (p=0.008), with NYHA class IV being more frequent among patients with sleep apnea (9.4% *vs.* 2.3%). Patients with sleep apnea had a greater burden of several major comorbidities, including hypertension (78.1% *vs.* 59.0%, p=0.003), diabetes mellitus (59.4% *vs.* 38.5%, p=0.001), atrial fibrillation/flutter (46.9% *vs.* 34.8%, p=0.047), and chronic obstructive pulmonary disease (34.4% *vs.* 14.2%, p<0.001). In contrast, the prevalence of coronary artery disease (53.1% *vs.* 57.8%, p=0.461), anemia (39.1% *vs.* 32.0%, p=0.234), iron deficiency (31.3% *vs.* 32.8%, p=0.798), valvular heart disease (20.3% *vs.* 17.5%, p=0.567), and previous cardiac surgery (25.0% *vs.* 22.7%, p=0.666) was similar between groups. Among patients with documented sleep apnea, only 43.8% were receiving positive airway pressure therapy.

**Conclusion:** Heart failure patients with documented sleep apnea had worse functional status and higher prevalences of hypertension, diabetes, atrial fibrillation/flutter, and COPD compared with patients without documented sleep apnea. Despite this greater comorbidity burden, fewer than half of patients with documented sleep apnea were receiving positive airway pressure therapy. These findings underscore the importance of greater awareness and appropriate evaluation of sleep apnea in patients with HF.

**Keywords:** Heart failure, sleep apnea, comorbidities, heart failure phenotype, functional status, positive airway pressure.

## [OP-28]

### Association between bundle branch block pattern and baseline clinical characteristics in patients with heart failure

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**Background:** Bundle branch block (BBB) is frequently encountered in patients with heart failure (HF) and may reflect differences in the underlying structural and clinical phenotype. We aimed to evaluate the association of bundle branch block patterns, particularly right bundle branch block (RBBB) and left bundle branch block (LBBB), with baseline clinical characteristics in patients with HF.

**Methods:** This cross-sectional analysis included patients followed in heart failure centers. Baseline demographic characteristics, HF phenotype and duration, functional status, cardiovascular comorbidities, previous interventions, implanted cardiac devices, and guideline-directed medical therapy were compared according to the presence of BBB and separately according to RBBB and LBBB patterns. Categorical variables were compared using the chi-square test.

**Results:** Among 2,222 patients with available baseline clinical and electrocardiographic data, 517 (23.3%) had a bundle branch block. RBBB was present in 162 (7.3%) and LBBB in 358 (16.1%) patients. Patients with BBB were more frequently male than those without BBB (67.3% *vs.* 61.7%; p=0.021).

HF phenotype differed significantly according to BBB status (p<0.001), with reduced ejection fraction HF being more frequent in patients with BBB (75.2% *vs.* 61.8%). This association was predominantly driven by LBBB: 83.0% of patients with LBBB had reduced ejection fraction HF compared with 61.4% of patients without LBBB (p<0.001), whereas HF phenotype did not differ significantly according to RBBB status (p=0.159). Functional status also differed according to BBB status (p=0.016), with a greater proportion of patients with BBB in higher NYHA functional classes. A similar difference was observed for LBBB (p=0.043). Coronary artery disease was more frequent in patients with BBB (62.3% *vs.* 56.5%; p=0.019), particularly in those with LBBB (64.8% *vs.* 56.5%; p=0.004), while no significant association was observed with RBBB. In contrast, atrial fibrillation/flutter was more frequent in patients with RBBB than in those without RBBB (42.6% *vs.* 33.0%; p=0.013), whereas it was less frequent among patients with LBBB (25.4% *vs.* 35.3%; p<0.001). Implanted cardiac devices were substantially more common in patients with BBB (41.2% *vs.* 23.3%; p<0.001), an association that was particularly pronounced in patients with LBBB (45.3% *vs.* 24.1%; p<0.001).

**Conclusion:** Bundle branch block identifies a distinct clinical phenotype among patients with heart failure. LBBB was strongly associated with reduced ejection fraction HF, greater functional impairment, coronary artery disease, and a higher prevalence of implanted cardiac devices, whereas RBBB was characterized by a higher prevalence of atrial fibrillation/flutter. These findings suggest that RBBB and LBBB represent clinically distinct conduction phenotypes within the heterogeneous HF population.

**Keywords:** Heart failure, bundle branch block, left bundle branch block, right bundle branch block, HFrEF, atrial fibrillation.

## [OP-29]

### Clinical profile and predictors of combined diuretic therapy in heart failure with stage 4 chronic kidney disease

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**Background:** Loop diuretic resistance is frequent in heart failure (HF) with advanced chronic kidney disease (CKD), and sequential nephron blockade is increasingly used. The profile of patients who actually receive combination diuretic therapy in this setting remains poorly characterized. We aimed to define this profile and its independent predictors in patients with HF and stage 4 CKD.

**Methods:** Ninety-four consecutive patients with HF and stage 4 CKD, who were admitted to our department between January 1, 2024, and June 30, 2025, were analyzed. Combination diuretic therapy was defined as a loop diuretic plus a second diuretic agent. The patients were divided into two groups based on the use of combined diuretic treatment. Groups were compared with chi-square and Mann-Whitney U tests. Independent predictors were identified by multivariable logistic regression analysis, including age, sex, hypertension, diabetes mellitus, atrial fibrillation, anemia, prior myocardial infarction, logBNP, logLVEF, and logeGFR.

**Results:** Combination diuretic therapy was used in 43 patients (45.7%); the second agent was acetazolamide in 23 (53.5%), an SGLT-2 inhibitor in 11 (25.6%) and a thiazide/thiazide-like diuretic in 9 (20.9%). Patients on combination therapy had a lower LVEF (40% [30-50] *vs.* 50% [40-55], p=0.039), markedly higher BNP (1746 [646–2747] *vs.* 644 [437-1013] pg/mL, p<0.001), and more frequent beta-blocker use (93.0% *vs.* 72.5%, p=0.010). Each one-unit increase in log BNP was associated with a 3.4-fold higher odds of receiving combination diuretic therapy (OR 3.449, 95% CI 1.682-7.072; p<0.001), and prior myocardial infarction status was the other independent predictor (OR 4.025, 95% CI 1.138-14.237; p=0.031) in HF with stage 4 CKD.

**Conclusion:** In HF with stage 4 CKD, nearly half of the patients received combination diuretic therapy, most often acetazolamide. Its use was driven by the increase in BNP level and ischemic heart disease rather than by the degree of renal impairment.

**Keywords:** Heart failure, stage 4 chronic kidney disease, combined diuretic therapy.

## [OP-30]

### Association between chronic digoxin use and early decongestion trajectory in patients with worsening HFrEF

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**Background:** Decongestion is a major therapeutic goal in patients hospitalized with worsening heart failure. Although the positive inotropic and neurohormonal effects of digoxin are well established, evidence regarding its effects on renal function and hemodynamics and their potential impact on natriuresis and diuretic response remains limited and inconsistent. We aimed to evaluate the association between chronic digoxin use and early decongestion trajectory in patients hospitalized with worsening HFrEF who were in sinus rhythm at admission.

**Methods:** This single-center retrospective cohort study included 140 patients hospitalized for worsening HFrEF between 2023 and 2026. Patients were classified according to chronic pre-admission digoxin use (digoxin, n=70; no digoxin, n=70). Congestion was assessed at baseline and at 24, 48, and 72 hours using the modified EVEREST composite congestion score (CCS; 0-9), based on orthopnea, jugular venous distention, and peripheral edema. The primary endpoint was the time-dependent change in CCS during the first 72 hours. Serial measurements were analyzed using a linear mixed-effects model, with the digoxin-by-time interaction assessing between-group differences. The adjusted model included age, body mass index, systolic blood pressure, LVEF, GFR, BNP, pre-admission furosemide-equivalent dose, and first-24-hour furosemide dose.

**Results:** Baseline EVEREST CCS was similar between groups (5.13±1.21 *vs.* 4.73±1.13; p=0.072). No significant differences were observed in LVEF, renal function, BNP, or pre-admission use of ACE inhibitors/ARBs/ARNIs, beta-blockers, MRAs, and SGLT2 inhibitors. First-24-hour furosemide dose (200 [200-240) *vs.* 200 [200-240] mg; p=0.724) and total in-hospital furosemide dose (1200 [1000-1800] *vs.* 1200 [1000-1800] mg; p=0.931) were also comparable. The reduction in EVEREST CCS from baseline to 72 hours was greater in the digoxin group (3.31±0.84 *vs.* 1.51±0.63 points; p<0.001). In the linear mixed-effects model, CCS declined significantly faster with digoxin (digoxin-by-time interaction: β=-0.579 points/24 h, 95% CI -0.639 to -0.518; p<0.001). This association remained significant after adjustment (β=-0.546 points/24 h, 95% CI -0.607 to -0.486; p<0.001). Among digoxin users, serum digoxin concentration was not correlated with 72-hour CCS reduction (Spearman ρ=0.080; p=0.511). Length of hospital stay was similar (7 [4-8] *vs.* 5 [5-8] days; p=0.331), whereas BNP reduction from admission to discharge was greater in the digoxin group (ΔBNP: 3419.5 [1869.8-10664.3] *vs.* 2214.0 [509.0-6109.5] pg/mL; p=0.008). In-hospital worsening renal function did not differ (10.0% *vs.* 8.6%; p=0.771). Ninety-day heart failure events were numerically lower with digoxin, but the difference was not statistically significant (4.3% *vs.* 14.3%; p=0.077).

**Conclusion:** Chronic digoxin use was associated with faster early decongestion in patients hospitalized with worsening HFrEF. This association and its potential underlying mechanisms should be confirmed in prospective studies.

**Keywords:** Digoxin, heart failure, systolic, natriuresis, diuretics, edema, cardiac.

## [OP-31]

### Pretreatment extracardiac manifestations and early response to Tafamidis in transthyretin amyloid cardiomyopathy

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**Background:** Transthyretin cardiac amyloidosis (ATTR-CM) is a progressive infiltrative cardiomyopathy frequently accompanied by extracardiac manifestations, including peripheral neuropathy (PN), carpal tunnel syndrome (CTS), and orthostatic hypotension (OH). Although these manifestations are recognized as important diagnostic clues, their influence on the early response to tafamidis therapy remains unclear. This study aimed to investigate whether pretreatment extracardiac manifestations are associated with early biochemical and functional response to tafamidis therapy.

**Methods:** Twenty-four consecutive patients with ATTR-CM who initiated tafamidis therapy between August 2023 and December 2025 were retrospectively analyzed. Baseline demographic, clinical, laboratory, and echocardiographic characteristics were recorded. Changes in N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels and 6-minute walk distance (6MWD) after six months of treatment were evaluated. Patients were further stratified according to the presence of PN, CTS, and OH to compare treatment responses between subgroups.

**Results:** The mean age was 73.1±10.4 years, and 50% of patients were female. The prevalence of PN, CTS, and OH was 54.2%, 45.8%, and 66.7%, respectively. After six months of tafamidis therapy, NT-proBNP levels decreased significantly (3488.0±3756.6 *vs.* 1609.6±1218.5 pg/mL, p=0.012), while 6MWD improved significantly (259.6±96.9 *vs.* 297.5±98.4 m, p=0.001), indicating an overall favorable early treatment response. No significant differences in NT-proBNP reduction, 6MWD improvement, or NYHA functional class improvement were observed according to PN or OH status. However, patients with CTS experienced significantly smaller improvements in 6MWD than those without CTS (Δ6MWD: 15.8±46.9 *vs.* 59.7±22.5 m, p=0.048). Although the proportion of patients achieving an improvement of at least one NYHA functional class was also lower in the CTS group (30.8% *vs.* 69.2%), this difference did not reach statistical significance (p=0.107). No significant between-group differences were observed regarding NT-proBNP changes.

**Conclusion:** Tafamidis therapy was associated with significant early improvements in both biochemical and functional parameters in patients with ATTR-CM, supporting its favorable short-term clinical effects. Although pretreatment peripheral neuropathy and orthostatic hypotension were not associated with differences in treatment response, patients with carpal tunnel syndrome demonstrated a smaller improvement in functional capacity after six months of therapy. While this finding should be interpreted cautiously given the limited sample size, it may reflect differences in disease phenotype or disease burden rather than a direct effect on treatment efficacy. These observations suggest that extracardiac manifestations, particularly carpal tunnel syndrome, may provide complementary clinical information when evaluating the early course of patients receiving tafamidis. Further prospective studies with larger cohorts are warranted to determine whether extracardiac manifestations have prognostic value for monitoring and predicting treatment response in ATTR-CM.

**Keywords:** Transthyretin cardiac amyloidosis, Tafamidis, extracardiac manifestations, carpal tunnel syndrome, treatment response.

## [OP-32]

### Non-atrial fibrillation risk factors associated with stroke in patients with heart failure

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**Background:** Patients with heart failure (HF) are at increased risk of ischemic stroke, with atrial fibrillation (AF) being a major established thromboembolic risk factor. However, stroke also occurs in patients with HF without AF, suggesting that additional vascular and metabolic factors contribute to cerebrovascular risk. This study aimed to identify clinical characteristics and cardiovascular risk factors associated with a history of stroke specifically among patients with HF without AF or atrial flutter.

**Methods:** This cross-sectional analysis included patients followed in HF centers. To specifically investigate non-AF-related stroke risk, all patients with AF or atrial flutter were excluded from the analysis. Patients without AF/flutter were classified according to the presence or absence of a history of stroke. Demographic characteristics, HF phenotype, functional status, hypertension, diabetes mellitus, smoking, renal function, coronary artery disease, peripheral artery disease, valvular heart disease, and other cardiovascular and systemic comorbidities were compared between groups.

**Results:** Among 1,495 patients with HF without AF/flutter, 105 (7.0%) had a history of stroke. Hypertension was more frequent among patients with previous stroke than among those without stroke (68.6% *vs.* 54.7%, p=0.023). Similarly, diabetes mellitus was more common in patients with previous stroke (50.5% *vs.* 36.3%, p=0.015). The strongest vascular association was observed for peripheral artery disease, which was present in 9.5% of patients with previous stroke compared with 3.6% of those without stroke (p=0.006). Coronary artery disease was also numerically more frequent in patients with previous stroke (69.5% *vs.* 60.3%), although the difference did not reach statistical significance (p=0.077). In contrast, the distribution of HF phenotype did not differ significantly according to stroke history (p=0.186), nor did sex, NYHA functional class, renal function category, valvular heart disease, pulmonary embolism, or anemia.

**Conclusion:** Stroke remains clinically relevant in patients with HF even in the absence of AF/flutter, affecting approximately 7% of this population. Hypertension, diabetes mellitus, and peripheral artery disease were significantly associated with previous stroke, highlighting the importance of the underlying vascular and metabolic risk burden independent of AF. These findings suggest that cerebrovascular risk assessment in HF should extend beyond rhythm status and incorporate systematic evaluation and management of conventional vascular risk factors.

**Keywords:** Heart failure, stroke, atrial fibrillation, hypertension, diabetes mellitus, peripheral artery disease, vascular risk.

## [OP-33]

### Clinical characteristics associated with guideline directed medical therapy in patients with heart failure

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**Background:** Despite substantial advances in guideline-directed medical therapy (GDMT) for heart failure (HF), implementation of all recommended treatment components remains suboptimal in routine clinical practice. Identifying clinical characteristics associated with the use of four-component GDMT may help define patient groups in whom treatment optimization is more challenging. This study aimed to compare baseline clinical characteristics between HF patients receiving four-component GDMT and those receiving fewer than four treatment components.

**Methods:** This cross-sectional analysis included HF patients followed in specialized heart failure centers. Guideline-directed therapy was assessed according to the use of four major treatment components, including a beta-blocker, renin-angiotensin system inhibition/angiotensin receptor-neprilysin inhibition, a mineralocorticoid receptor antagonist, and a sodium-glucose cotransporter-2 inhibitor. Patients receiving all four drug classes were classified as the four-component GDMT group, whereas those receiving zero to three classes were classified as the 0-3 component GDMT group. Baseline demographic characteristics, HF phenotype and duration, NYHA functional class, comorbidities, renal function, cardiovascular history, and previous hospitalizations were compared between groups.

**Results:** Among 2,230 patients with complete clinical data, 677 (30.4%) were receiving four-component GDMT, whereas 1,553 (69.6%) were receiving fewer than four treatment components. Patients receiving four-component GDMT were more frequently male (70.0% *vs.* 60.0%; p<0.001). HF phenotype differed significantly between groups (p<0.001). HFrEF was more frequent among patients receiving four-component GDMT (86.1% *vs.* 55.8%), whereas HFpEF was less frequent (7.5% *vs.* 32.5%). NYHA functional class also differed significantly between groups (p<0.001), with NYHA class I being more frequent (24.1% *vs.* 16.9%) and class III less frequent (22.9% *vs.* 27.7%) among patients receiving four-component GDMT. Coronary artery disease (64.1% *vs.* 55.6%; p<0.001), diabetes mellitus (43.3% *vs.* 36.8%; p=0.004), and implanted cardiac devices (34.6% *vs.* 24.5%; p<0.001) were more frequent among patients receiving four-component GDMT. In contrast, Atrial fibrillation/flutter was less frequent among patients receiving four-component GDMT (29.2% *vs.* 35.9%; p=0.003). Renal function distribution differed markedly between groups (p<0.001), with advanced renal dysfunction categories being substantially less frequent among patients receiving four-component GDMT. Previous HF hospitalization was more frequent in patients receiving optimal therapy (41.2% *vs.* 34.5%; p=0.035). Malignancy was numerically less frequent in the optimal therapy group (4.6% *vs.* 6.5%), although this difference was not statistically significant (p=0.094).

**Conclusion:** Only approximately one-third of patients followed in heart failure centers were receiving complete four-component GDMT. Achievement of optimal therapy was strongly associated with HFrEF phenotype, male sex, better renal function, coronary artery disease, and the presence of an implanted cardiac device. Conversely, patients with preserved ejection fraction, advanced renal dysfunction, and atrial fibrillation were less likely to receive all four treatment components.

**Keywords:** Heart failure, guideline-directed medical therapy, optimal medical therapy, HFrEF, renal function, treatment optimization.

## [OP-34]

### Lower guideline-directed medical therapy intensity in heart failure patients with concomitant malignancy

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**Background:** Patients with malignancy and heart failure (HF) may experience barriers to the implementation of guideline-directed medical therapy (GDMT), including drug interactions, renal or electrolyte concerns, and competing clinical priorities during cancer treatment. This multicenter study aimed to compare the use and overall intensity of guideline-recommended HF therapy between patients with and without malignancy.

**Methods:** A total of 2,230 patients with available treatment and malignancy data were included and categorized according to the presence of malignancy into malignancy (n=132) and non-malignancy (n=2,098) groups. The use of beta-blockers, renin-angiotensin system inhibitors (RASi; ACE inhibitors, ARBs, or ARNIs), mineralocorticoid receptor antagonists (MRAs), and sodium-glucose cotransporter-2 inhibitors (SGLT2is) was evaluated. The number of guideline-directed treatment components was calculated as an overall medical therapy score ranging from 0 to 4. Categorical variables were compared using the chi-square test. A p-value <0.05 was considered statistically significant.

**Results:** In the overall HF cohort, 62.9% of patients were male. HFrEF was the predominant HF phenotype (64.9%), followed by HFpEF (25.1%) and

HFmrEF (10.0%). Most patients were in NYHA functional class II (48.1%) or III (26.3%). Hypertension was present in 59.7%, diabetes mellitus in 38.6%, coronary artery disease in 58.3%, atrial fibrillation/flutter in 33.8%, chronic obstructive pulmonary disease in 14.8%, and anemia in 31.6%. Among the 2,230 patients included in the treatment analysis, beta-blocker use was 87.9% in patients with malignancy compared with 90.6% in those without malignancy (p=0.310). Similarly, no significant differences were observed for ACE inhibitor (43.9% *vs.* 46.7%, p=0.543), ARB (17.4% *vs.* 18.6%, p=0.738), ARNI (9.1% *vs.* 11.3%, p=0.426), or SGLT2 inhibitor use (41.7% *vs.* 48.5%, p=0.129). In contrast, MRA use was significantly lower among patients with malignancy (46.2% *vs.* 62.1%, p<0.001). The overall distribution of the number of GDMT components differed significantly between patients with and without malignancy ($\chi^2$=12.546, p=0.014), with a significant trend toward fewer treatment components among patients with malignancy (linear-by-linear association, p=0.002). Three or four GDMT components were used in only 50.0% of patients with malignancy compared with 64.6% of those without malignancy (p<0.001). Complete four-component therapy was also numerically less frequent in patients with malignancy (23.5% *vs.* 30.8%).

**Conclusion:** In this multicenter cross-sectional study, patients with concomitant malignancy and HF received fewer components of GDMT than patients without malignancy. The difference was particularly pronounced for MRA use, while the use of other individual drug classes did not differ significantly. Only half of patients with malignancy received three or four GDMT components, compared with nearly two-thirds of patients without malignancy. These findings suggest a clinically relevant treatment gap in HF patients with malignancy and highlight the importance of closer cardio-oncology collaboration to identify and address barriers limiting GDMT optimization in this population.

**Keywords:** Malignancy, heart failure, guideline-directed medical therapy, cardio-oncology, treatment optimization, multicenter.

## [OP-35]

### Elevated hs-CRP identifies a high-risk heart failure phenotype: A multicenter cross-sectional analysis

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**Background:** High-sensitivity C-reactive protein (hs-CRP) is a systemic inflammation marker that may identify heart failure (HF) patients with greater disease burden. We aimed to evaluate the association between elevated hs-CRP and HF phenotype, functional status, comorbidities, healthcare utilization, and medical therapy in a multicenter cohort.

**Methods:** This cross-sectional study included HF patients with hs-CRP measurements at enrollment, categorized as hs-CRP ≤5 mg/L or >5 mg/L. Baseline demographics, HF phenotype, NYHA class, hospitalizations, comorbidities, renal function, and guideline-directed medical therapy (GDMT) were compared between groups using chi-square tests (p<0.05 considered significant).

**Results:** Among 1,120 included patients, 545 had hs-CRP ≤5 mg/L and 575 had hs-CRP >5 mg/L. HF phenotype differed significantly (p=0.008); HFrEF was less frequent in the elevated hs-CRP group (69.2% *vs.* 76.9%), while HFpEF was more frequent (22.3% *vs.* 15.2%). NYHA class III was significantly more common in the elevated hs-CRP group (35.0% *vs.* 20.6%, p<0.001). Elevated hs-CRP was associated with greater healthcare utilization, including previous HF hospitalizations (45.5% *vs.* 29.5%, p<0.001), emergency visits (48.9% *vs.* 31.4%, p<0.001), and need for oral diuretic dose escalation (28.3% *vs.* 18.0%, p<0.001). These patients also had higher prevalences of hypertension (61.4% *vs.* 51.2%, p=0.001), diabetes (43.0% *vs.* 32.5%, p<0.001), atrial fibrillation/flutter (36.7% *vs.* 25.7%, p<0.001), anemia (38.1% *vs.* 23.5%, p<0.001), and valvular heart disease (16.2% *vs.* 10.5%, p=0.005). Renal function distribution differed markedly (p<0.001), with advanced dysfunction (categories 3-5) accounting for 28.1% of the elevated hs-CRP group compared with 12.1% of the lower group. Regarding GDMT, ACE inhibitor (44.4% *vs.* 51.3%, p=0.022), ARB (13.3% *vs.* 20.8%, p=0.001), and MRA use (61.4% *vs.* 69.3%, p=0.006) were lower in patients with elevated hs-CRP, whereas SGLT2 inhibitor use was higher (50.6% *vs.* 40.3%, p=0.001). However, complete four-component GDMT use was virtually identical between groups (29.7% *vs.* 29.4%, p=0.906).

**Conclusion:** Elevated hs-CRP identifies a distinct, clinically vulnerable HF phenotype with worse functional status, greater renal dysfunction, higher comorbidity burden, and increased healthcare utilization. Despite differences in individual drug classes, overall four-component GDMT use remained similar. hs-CRP serves as a simple, accessible marker of increased clinical burden in HF.

**Keywords:** Heart failure, high-sensitivity C-reactive protein, hs-CRP, biomarker, systemic inflammation, heart failure phenotype, healthcare utilization, comorbidities.

## [OP-36]

### Prevalence and patterns of mixed valvular heart disease in patients with heart failure

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**Background:** Mixed valvular heart disease (VHD) is frequently encountered in patients with heart failure (HF), yet its distribution across different left ventricular ejection fraction (LVEF) phenotypes remains insufficiently characterized. This study aimed to determine the prevalence and patterns of mixed VHD in patients with HF and to evaluate its association with HFrEF, HFmrEF, and HFpEF phenotypes.

**Methods:** This cross-sectional analysis included patients followed in HF centers. Moderate-to-severe aortic stenosis, aortic regurgitation, mitral stenosis, mitral regurgitation, pulmonary stenosis, tricuspid regurgitation, and tricuspid stenosis were evaluated. Mixed VHD was defined as the coexistence of at least two moderate-to-severe valvular lesions. The prevalence and patterns of mixed VHD were assessed, and its distribution was compared across HFrEF, HFmrEF, and HFpEF phenotypes.

**Results:** Among 2,259 patients with available LVEF phenotype and valvular data, 1,466 (64.9%) had HFrEF, 227 (10.0%) had HFmrEF, and 566 (25.1%) had HFpEF. Overall, mixed VHD was present in 715 patients (31.7%). The prevalence of mixed VHD differed significantly across HF phenotypes ($p<0.001$), being substantially higher in patients with HFpEF (40.8%) than in those with HFrEF (28.8%) or HFmrEF (27.3%). HFrEF and HFmrEF showed similar rates of mixed VHD, whereas HFpEF was characterized by a distinctly greater burden of multivalvular involvement. Among patients with mixed VHD, concomitant moderate-to-severe mitral and tricuspid regurgitation represented the predominant valvular pattern, while other multivalvular combinations were considerably less frequent.

**Conclusion:** Mixed valvular heart disease was present in nearly one-third of patients followed in HF centers and showed a strong association with LVEF phenotype. The burden was particularly pronounced in HFpEF, affecting more than 40% of these patients, compared with approximately 28% of patients with HFrEF or HFmrEF. These findings identify mixed VHD as an important component of the HFpEF phenotype and emphasize the importance of comprehensive multivalvular assessment in patients with HF, particularly those with preserved LVEF.

**Keywords:** Heart failure, mixed valvular heart disease, HFpEF, HFrEF, mitral regurgitation, tricuspid regurgitation, multivalvular disease.

## [OP-37]

### Suboptimal vaccination coverage in heart failure patients: A multicenter cross-section

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**Background:** Vaccination represents an important component of preventive care in patients with heart failure (HF), who are particularly vulnerable to complications of vaccine-preventable infections. This multicenter study aimed to evaluate influenza, pneumococcal, COVID-19, and herpes zoster vaccination coverage among patients followed in HF centers.

**Methods:** This cross-sectional study included 2,272 patients with HF. Vaccination status for influenza, COVID-19, pneumococcal, and herpes zoster vaccines was assessed using hospital and national health records. Vaccination status was classified as vaccinated, unvaccinated, or unknown, where applicable. Baseline demographic and clinical characteristics were recorded. Vaccination coverage was additionally calculated among patients with known vaccination status for vaccines with an unknown-status category.

**Results:** A total of 2,272 patients with HF were included, of whom 2,259 had complete baseline clinical data. Among patients with available data, 62.9% were male. HFrEF was the predominant HF phenotype (64.9%), followed by HFpEF (25.1%) and HFmrEF (10.0%). Most patients were in NYHA functional class II (48.1%) or III (26.3%). Hypertension was present in 59.7%, diabetes mellitus in 38.6%, coronary artery disease in 58.3%, atrial fibrillation/flutter in 33.8%, chronic obstructive pulmonary disease in 14.8%, and anemia in 31.6%. Influenza vaccination status was available for 2,259 patients; 458 (20.3%) were vaccinated, 1,274 (56.4%) were unvaccinated, and vaccination status was unknown in 527 (23.3%). Among patients with known vaccination status, influenza vaccination coverage was 26.4% (458/1,732). Similarly, 493 (21.8%) patients had received pneumococcal vaccination, 1,236 (54.7%) were unvaccinated, and vaccination status was unknown in 530 (23.5%); among patients with known status, pneumococcal vaccination coverage was 28.5% (493/1,729). COVID-19 vaccination coverage was substantially higher, with 1,856 of 2,259 patients (82.2%) vaccinated. In contrast, herpes zoster vaccination was exceptionally rare; among 1,093 patients with available data, only 3 (0.3%) had been vaccinated.

**Conclusion:** Vaccination coverage among patients followed in HF centers was suboptimal and markedly heterogeneous across vaccine types. Although COVID-19 vaccination coverage exceeded 80%, only approximately one-quarter of patients with known vaccination status had received influenza or pneumococcal vaccination, while herpes zoster vaccination was exceptionally rare. Moreover, vaccination status was unknown in nearly one-quarter of patients for influenza and pneumococcal vaccines, highlighting important gaps in systematic vaccination assessment and documentation. Integrating structured vaccination assessment and appropriate immunization strategies into routine HF care may represent an important opportunity to strengthen preventive care in this high-risk population.

**Keywords:** Heart failure, vaccination, influenza, pneumococcal vaccination, COVID-19, herpes zoster, preventive care, multicenter.

## [OP-38]

### Clinical characteristics of heart failure patients aged <40 years

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**Background:** Heart failure (HF) in young adults represents a distinct clinical phenotype in which traditional cardiovascular comorbidities may be less prominent and non-ischemic or genetic etiologies may play a greater role. We aimed to characterize HF patients aged <40 years and compare their clinical characteristics with older HF patients.

**Methods:** This cross-sectional analysis included patients from a large HF cohort. Patients were categorized according to age as <40 years and ≥40 years. Demographic characteristics, HF phenotype, functional status, comorbidities, previous cardiovascular interventions, and guideline-directed medical therapy were compared between groups using chi-square tests. Of 2,161 patients with available age-group data, 179 (8.3%) were aged <40 years and 1,982 (91.7%) were aged ≥40 years.

**Results:** Sex distribution was similar between young and older patients (male: 65.9% *vs.* 63.2%, p=0.472). HFrEF was substantially more frequent among patients aged <40 years (83.8% *vs.* 63.7%), whereas HFpEF was less frequent (7.3% *vs.* 26.2%; p<0.001). Young patients also had a more favorable functional profile, with NYHA class I observed in 39.1% *vs.* 17.8%, while NYHA class III-IV symptoms were considerably less frequent (p<0.001). Traditional cardiovascular comorbidities were markedly less prevalent in young patients. Hypertension was present in 18.4% *vs.* 63.7% (p<0.001) and diabetes mellitus in 11.2% *vs.* 41.6% (p<0.001). Renal dysfunction was also substantially less common among younger patients (p<0.001). In contrast, genetic cardiomyopathy was markedly enriched in the young HF population (10.1% *vs.* 1.1%, p<0.001). Young patients had fewer emergency department visits (25.7% *vs.* 40.4%, p<0.001) and less frequent need for oral diuretic dose escalation (10.1% *vs.* 22.9%, p<0.001), whereas hospitalization rates did not differ significantly (34.7% *vs.* 36.9%, p=0.718). Regarding HF therapy, younger patients more frequently received beta-blockers (95.5% *vs.* 90.4%, p=0.025), ACE inhibitors (59.6% *vs.* 45.7%, p<0.001), and mineralocorticoid receptor antagonists (78.1% *vs.* 60.3%, p<0.001), but less frequently received SGLT2 inhibitors (34.3% *vs.* 49.5%, p<0.001).

**Conclusion:** Patients with HF aged <40 years constitute a distinct HF phenotype characterized by a striking predominance of HFrEF despite substantially lower burdens of hypertension, diabetes, and renal dysfunction. Genetic cardiomyopathy was markedly more prevalent in this population, supporting a greater contribution of non-traditional and inherited etiologies to HF at younger ages. Despite the high prevalence of reduced LVEF, young patients had better functional status and fewer emergency visits and required less diuretic intensification. These findings emphasize the importance of etiologic, particularly genetic, evaluation in young adults presenting with HF.

**Keywords:** Heart failure, young adults, genetic cardiomyopathy, heart failure phenotype, reduced ejection fraction, traditional comorbidities.

## [OP-39]

### Relationship between right ventricular fractional area change and renal function in non-dialysis chronic kidney disease

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**Background:** The aim of this study was to evaluate the relationship between renal function and right ventricular (RV) structure and function in a non-dialysis chronic kidney disease (CKD) population selected with strict cardiac and systemic exclusion criteria. Considering that RV fractional area change (RVFAC) is an echocardiographic parameter sensitive to changes in RV volume, geometry, and loading conditions rather than a direct indicator of myocardial contractility, the relationship between eGFR levels and RVFAC was examined in order to demonstrate subclinical geometric and functional RV adaptations in non-dialysis CKD.

**Methods:** A total of 300 non-dialysis CKD patients with an estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m$^2$ were included. Patients with a history of dialysis or kidney transplantation, moderate or severe valvular disease, prosthetic valves, coronary artery disease or revascularization, LVEF <50%, heart failure, hypertrophic or infiltrative cardiomyopathy, pulmonary embolism, or chronic constrictive pericarditis were excluded. Patients were classified as G3a (eGFR 45-59 mL/min/1.73 m$^2$; n=128), G3b (30-44; n=97), and G4-5 (<30; n=75); because only 6% (n=18) had an eGFR <15 mL/min/1.73 m$^2$, stages G4 and G5 were combined. RV structure and function were assessed at rest by two-dimensional (2D) and strain echocardiography and compared between the groups.

**Results:** The mean age was 63.4±12.2 years; 59.3% of the patients were female (n=178). Hypertension was present in 86% (n=258), diabetes mellitus in 38% (n=114), hyperlipidemia in 29% (n=86), and atrial fibrillation in 3.3% (n=10). RV structural and functional parameters—right atrial area and diameter, RV end-diastolic and end-systolic areas, RV free wall strain (RVFWSL), RV global strain (RV4CSL), RV basal, mid, and longitudinal diameters, TAPSE, SPAP, lateral s′ velocity, and tricuspid jet velocity—were within normal reference ranges and showed no significant differences between the eGFR groups (p>0.05 for all comparisons). In contrast, RVFAC (44.8±9.4% in the entire cohort) differed significantly between the groups: 43.1±8.5% in G3a, 45.8±9.7% in G3b, and 46±10% in G4-5 (p=0.043). Post-hoc analyses revealed significant differences

between G3a and G3b (p=0.037) and between G3a and G4-5 (p=0.035), but not between G3b and G4-5 (p=0.878). In correlation analyses, eGFR correlated negatively with RVFAC (rho=-0.179, p=0.007), whereas serum creatinine correlated positively with RVFAC (rho=0.167, p=0.013). These findings indicate a significant increase in RVFAC as eGFR declines.

**Conclusion:** In non-dialysis CKD patients without clinically overt cardiovascular disease, RV structure and function were generally preserved, whereas RVFAC increased significantly in parallel with the decline in renal function. This finding supports the view that RVFAC is more sensitive to changes in ventricular geometry and filling conditions than to myocardial contractility, and suggests that the RV may exhibit subclinical geometric adaptation to hemodynamic loading before changes appear in conventional right heart function parameters. RVFAC may therefore be considered a potential echocardiographic marker of early RV adaptation in non-dialysis CKD patients.

**Keywords:** Chronic kidney disease, right ventricular fractional area change, right ventricular adaptation, echocardiography.

## [OP-40]

### From a hypertrophic phenotype to biventricular failure in Danon disease: VA-ECMO support while awaiting transplantation

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**Background:** Danon disease is a rare X-linked dominant lysosomal storage disorder caused by loss-of-function LAMP2 variants. In affected males, hypertrophic cardiomyopathy, arrhythmias, conduction disease, and rapidly progressive heart failure may appear early in life. We report a young man who progressed to advanced biventricular failure.

**Case Report:** A 26-year-old man diagnosed with cardiomyopathy at age 17 received an ICD in 2016. Echocardiography performed at another center in 2023 showed preserved LVEF; however, subsequent deterioration in systolic function prompted a CRT-D upgrade in 2024. He had paroxysmal atrial fibrillation, atrial tachycardia, and repeated admissions for decompensated heart failure in 2024. In February 2025, BNP was 5,170 pg/mL; echocardiography showed LVEF 29%, LV end-diastolic diameter of 4.8 cm, septal and posterior wall thickness 1.6 cm, and global hypokinesia. A 2020 chest CT demonstrated marked left ventricular hypertrophy. The right ventricle showed severe apical and mid-lateral hypokinesia with microaneurysms and a dilated, thinned outflow tract. There was moderate tricuspid regurgitation, SPAP of 37 mmHg, and a 1.8-cm pericardial effusion. Catheterization showed a cardiac index of 2.52 L/min/m$^2$ and PVR of 3.42 Wood units. Genetic testing, phenotype, and family evaluation confirmed Danon disease. Assist device implantation was considered unfeasible because of the small, nondilated left ventricular cavity, and escalating inotropic requirements led to urgent transplant listing in March 2025. By July 2025, LVEF had fallen to 25% and the effusion had increased to 3.2 cm without tamponade. In October 2025, recurrent syncope from loss of ventricular capture required temporary pacing and CRT-D lead revision. From December 2025, persistent hypotension required increasing vasopressor and inotropic support. In December, blood cultures grew MRSA; transthoracic imaging was unremarkable, but transesophageal echocardiography showed a mobile 13-mm vegetation on the lead at the tricuspid valve. Extraction was not performed because he was pacing-dependent and his anatomy precluded lead reimplantation; he completed a 42-day course of cefazolin and teicoplanin with sterile cultures and clinical improvement. In February 2026, he was intubated for rising lactate and hemodynamic instability after pericardiocentesis; VA-ECMO was initiated and his urgency upgraded to status 1A. He required renal replacement therapy and died after cardiac arrest with sepsis on February 20, 2026.

**Discussion:** In Danon disease, a hypertrophied but non-dilated left ventricle may progress to advanced disease with reduced LVEF. Right ventricular involvement, pericardial effusion, and conduction abnormalities requiring device therapy may accompany deterioration, and recurrent decompensation with vasopressor and inotrope dependence underscores the aggressive course. When assist device support is not feasible, transplantation should be considered before multiorgan failure.

**Conclusion:** Danon disease should be considered in young men with early-onset hypertrophic cardiomyopathy accompanied by arrhythmias or conduction abnormalities, systolic dysfunction, or recurrent heart failure admissions. Genetic testing and family screening should be performed early, and referral to an advanced heart failure and transplant center should not be delayed, as deterioration can be rapid and the transplantation window may close quickly.

**Keywords:** Danon disease, burned-out hypertrophic cardiomyopathy, extracorporeal membrane oxygenation, infective endocarditis, pericardial effusion.

## [OP-41]

### Cardiac sarcoidosis or genetic cardiomyopathy? Severe biventricular failure in a young patient

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**Background:** Isolated cardiac sarcoidosis can present with a phenotype that overlaps with inflammatory and genetic cardiomyopathies. When histologic confirmation is difficult to obtain, multimodality imaging can guide the diagnostic work-up but is not definitive on its own. We describe the diagnostic challenge in a young patient with severe biventricular failure and ventricular arrhythmia.

**Case Report:** A 28-year-old man was diagnosed with heart failure in November 2025 and referred to our center in January 2026 for heart transplantation/left ventricular assist device evaluation. He was born to consanguineous parents, but there was no known family history of cardiomyopathy. At presentation, BNP was 961 pg/mL. The admission ECG showed frequent PVCs in a bigeminal pattern. Echocardiography demonstrated an LVEF of 10-15%, severe global hypokinesia, biventricular systolic dysfunction, moderate-to-severe tricuspid regurgitation, and pericardial effusion. Speckle-tracking analysis showed a left ventricular global longitudinal strain of -6.0%. CMR showed an LVEF of 16% and an RVEF of 14%, with septal-predominant ring-like mid-myocardial late gadolinium enhancement, native T1 values of 1250-1300 ms, T2 of 72 ms, an extracellular volume fraction of 37%. Ambulatory ECG monitoring recorded 12,127 isolated PVCs and an episode of wide-complex tachycardia at 132 beats/min. FDG-PET/CT showed biventricular and atrial uptake, most prominent in the lateral-inferolateral left ventricular wall, without pathologic pulmonary or mediastinal uptake. Core-needle biopsy of an FDG-avid right axillary lymph node showed reactive changes, and chest CT showed no evidence of pulmonary sarcoidosis. Prednisolone and mycophenolate mofetil were started for presumed isolated cardiac sarcoidosis. Heart failure therapy was optimized, and an ICD was implanted in March 2026. Initial genetic testing covering MYBPC3, TNNT2, TPM1, MYH6, MYH7, TNNI3, and MYL2 was negative. A second consultation with the medical genetics team was requested to assess possible variants in DSP, FLNC, and other cardiomyopathy-associated genes; the expanded analysis remains pending. Severe left ventricular dysfunction persisted in June 2026 (LVEF 10.4%). The patient was readmitted with congestion in August. A 2.3x2.4 cm immobile thrombus was detected at the right ventricular apex around the ICD lead; Evaluation for LVAD/BiVAD support is ongoing.

**Discussion:** Elevated T2 and extracellular volume on CMR, together with focal myocardial FDG uptake, support active inflammation. However,

the ring-like mid-myocardial fibrosis, substantial arrhythmic burden, parental consanguinity, absence of extracardiac granulomatous disease all reduce diagnostic certainty. DSP or FLNC associated with arrhythmogenic cardiomyopathy therefore remains an important phenocopy. The absence of improvement in systolic function despite immunosuppressive therapy suggests an advanced phenotype in which diffuse fibrosis, rather than active inflammation, may be dominant.

**Conclusion:** In young patients with arrhythmic biventricular cardiomyopathy, the distinction between cardiac sarcoidosis and genetic or inflammatory cardiomyopathy should not be reduced to a binary interpretation of imaging findings alone. Targeted tissue sampling, and prompt genetic testing, with broader analysis when indicated, are critical to the timing of immunosuppression, ICD implantation, and advanced heart failure therapies.

**Keywords:** Cardiac sarcoidosis, genetic cardiomyopathy, biventricular heart failure.

## [OP-42]

### Naples prognostic score as a predictor of 1-year mortality in HFrEF patients hospitalized for acute decompensated HF

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**Background:** Heart failure with reduced ejection fraction (HFrEF) is a global health concern associated with significant morbidity, mortality, and healthcare burden. Despite advances in pharmacological and device-based therapies, patients with HFrEF continue to face high risks of hospitalization and death, both in the short and long term. Malnutrition and increased inflammation are major factors associated with HFrEF progression and poor prognosis. The Naples prognostic score (NPS), comprising serum albumin, total cholesterol, neutrophil-to-lymphocyte ratio, and lymphocyte-to-monocyte ratio, enables integrated assessment of inflammatory and nutritional status. This study aimed to evaluate the association of NPS with 1-year all-cause mortality and its prognostic value in patients with HFrEF hospitalized for acute decompensation.

**Methods:** A total of 246 patients with HFrEF hospitalized for acute decompensated heart failure were included. NPS was calculated from admission laboratory data, and patients were classified into NPS 0-2 points (n=79) and NPS 3-4 points (n=167) groups. The primary endpoint was 1-year all-cause mortality. Demographic, clinical, laboratory, and echocardiographic characteristics were compared between groups. Survival was assessed using Kaplan-Meier analysis and the log-rank test. The association between NPS and mortality was evaluated by Cox regression, and prognostic performance by ROC analysis.

**Results:** During 1 year of follow-up, all-cause mortality occurred in 63 patients (25.6%): 13 (16.46%) in the low-NPS group and 50 (29.94%) in the high-NPS group (p=0.024). Age and the prevalence of diabetes mellitus, coronary artery disease, chronic kidney disease, permanent atrial fibrillation, and ischemic etiology were significantly higher in the high-NPS group (all $p<0.05$). Kaplan-Meier analysis showed significantly lower survival in the high-NPS group (log-rank p=0.035). In multivariable Cox regression, each 1-point increase in NPS was independently associated with a 37% higher risk of 1-year all-cause mortality (aHR 1.37, 95% CI 1.041.82; p=0.026). ROC analysis yielded an AUC of 0.612 (95% CI 0.538-0.687; p=0.003). The optimal NPS cut-off for mortality was 4, with 44.7% sensitivity and 71.5% specificity.

**Discussion:** NPS was independently associated with 1-year all-cause mortality in patients with acutely decompensated HFrEF, and higher NPS values were associated with poorer survival. However, the discriminative performance of NPS alone was limited.

**Conclusion:** As an easily calculated score derived from routine laboratory parameters, NPS may have value as a complementary prognostic marker for risk stratification in patients with HFrEF.

**Keywords:** Heart failure, reduced ejection fraction, inflammation, malnutrition, mortality.

## [OP-43]

### RV-PA coupling is associated with exercise capacity beyond left ventricular ejection fraction in heart failure

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**Background and Aim:** Right ventricular-pulmonary arterial (RV-PA) coupling, estimated by the ratio of tricuspid annular plane systolic excursion to systolic pulmonary artery pressure (TAPSE/sPAP), integrates right ventricular contractility and afterload. Because left ventricular ejection fraction (LVEF) correlates poorly with functional capacity in heart failure, we examined whether TAPSE/sPAP was independently associated with cardiopulmonary exercise test (CPET) performance.

**Methods:** Patients with chronic heart failure who underwent echocardiography and symptom-limited CPET were analyzed retrospectively. Paired TAPSE/sPAP and peak $VO_2$ data were available in 228 patients and paired TAPSE/sPAP and $VE/VCO_2$ slope data in 234; complete-case multivariable models included 194 and 200 patients, respectively. Associations were assessed using Spearman correlation and linear regression adjusted for age, sex, LVEF, ln(BNP+1), hemoglobin, eGFR, and body mass index. ROC analysis evaluated TAPSE/sPAP for peak $VO_2$ <14 and <12 mL/kg/min and $VE/VCO_2$ slope >34.

**Results:** Among 370 patients (median age 51 years; 27.0% women; median LVEF 33%), median TAPSE/sPAP was 0.80. TAPSE/sPAP correlated with peak $VO_2$ ($r=0.297$, $p<0.001$) and $VE/VCO_2$ slope ($r=-0.393$, $p<0.001$). It remained independently associated with higher peak $VO_2$ (standardized $\beta=0.161$, $p=0.014$; $R^2=0.399$) and lower $VE/VCO_2$ slope (standardized $\beta=-0.227$, $p=0.002$; $R^2=0.254$), whereas LVEF was not independently associated with either endpoint (p=0.501 and p=0.081). Discrimination was modest for peak $VO_2$ <14 mL/kg/min (AUC 0.617) and acceptable for peak $VO_2$ <12 mL/kg/min (AUC 0.709; cut-off ≤0.76). Performance was strongest for $VE/VCO_2$ slope >34 (AUC 0.720; cut-off ≤0.73; sensitivity 60.8%, specificity 80.3%).

**Discussion:** Higher TAPSE/sPAP was associated with better aerobic capacity and ventilatory efficiency, suggesting that exercise limitation may be more closely related to right ventricular adaptation to pulmonary arterial load than to resting LVEF alone. TAPSE/sPAP is a validated non-invasive surrogate of RV-PA coupling, and impaired RV-PA coupling provides prognostic information across heart failure phenotypes. Its stronger association with $VE/VCO_2$ slope may reflect the effects of pulmonary vascular load and impaired pulmonary perfusion on ventilatory efficiency. However, its moderate discriminatory performance indicates that it should complement rather than replace CPET.

**Conclusion:** TAPSE/sPAP was independently associated with aerobic capacity and ventilatory efficiency and may provide a simple echocardiographic marker of exercise limitation beyond conventional LVEF assessment.

**Keywords:** Heart failure, RV-PA coupling, TAPSE/sPAP, cardiopulmonary exercise testing, peak $VO_2$, $VE/VCO_2$ slope.

## [OP-44]

**Best Oral Presentation Candidate**

### Obesity and left ventricular outflow tract obstruction in patients with a hypertrophic LV phenotype

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**Background:** Obesity is a modifiable cardiovascular risk factor affecting cardiac structure and function. However, data on its association with left ventricular outflow tract obstruction (LVOTO) in patients with a hypertrophic left ventricular phenotype are limited. This study evaluated the association of obesity with LVOTO, echocardiographic findings, and clinical characteristics in this population.

**Methods:** This prospective observational study included 280 patients with an interventricular septal thickness ≥15 mm. Patients were categorized as non-obese (BMI <30 kg/m$^2$, n=191) or obese (BMI ≥30 kg/m$^2$, n=89). LVOT gradients were assessed at rest and during physiological provocation using continuous-wave Doppler echocardiography. LVOTO was defined as a peak instantaneous LVOT gradient ≥30 mmHg at rest or with provocation. Clinical, echocardiographic, and biochemical parameters were compared between groups. Factors independently associated with LVOTO were assessed using multivariable logistic regression.

**Results:** Of 280 patients, 89 (31.8%) were obese. Female sex was more frequent in the obese group (46.1% *vs.* 28.8%; p=0.005). LVOTO was significantly more prevalent among obese patients (24.7% *vs.* 14.1%; p=0.030), and the provoked LVOT gradient was higher (10 [6-37] *vs.* 7 [5-12.5] mmHg; p=0.003). Resting LVOT gradient, maximum wall thickness, left atrial volume index, left ventricular mass index, left atrial reservoir strain, LV global longitudinal strain, and NT-proBNP levels did not differ significantly between groups. In multivariable analysis, obesity (OR=2.015; 95% CI: 1.051-3.861; p=0.035) and diabetes mellitus (OR=2.772; 95% CI: 1.116-6.888; p=0.028) were independently associated with LVOTO. BMI showed a weak positive correlation with provoked LVOT gradient (r=0.145; p=0.015) and a weak negative correlation with NT-proBNP (r=-0.171; p=0.004).

**Conclusion:** Obesity was independently associated with a higher prevalence of LVOTO and higher provoked LVOT gradients in patients with a hypertrophic left ventricular phenotype. Despite similar maximum wall thickness and left ventricular mass index, the greater frequency of obstruction among obese patients suggests that this association may not be explained solely by the degree of hypertrophy. Obesity may therefore represent a modifiable clinical factor associated with obstructive physiology in this population.

**Keywords:** Obesity, hypertrophic cardiomyopathy, left ventricular outflow tract obstruction, body mass index, echocardiography.

## [OP-45]

***Best Oral Presentation Candidate***

### The prognostic role of left atrial deformation in patients with moderate aortic stenosis

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**Background:** Aortic stenosis is a progressive valvular heart disease associated with structural and functional changes in both the left ventricle and left atrium. While the management of severe aortic stenosis is well established, risk stratification in patients with moderate aortic stenosis remains challenging. Left atrial deformation assessed by speckle-tracking echocardiography may provide additional information regarding the hemodynamic consequences of aortic stenosis before progression to severe disease. This study aimed to investigate the prognostic value of left atrial strain parameters in patients with moderate aortic stenosis and their association with adverse clinical outcomes.

**Methods:** Patients with moderate aortic stenosis at the time of echocardiographic evaluation were retrospectively included. Echocardiographic images were analyzed, and left atrial reservoir, conduit, and contraction strain parameters were assessed using speckle-tracking echocardiography. Patient medical histories and outpatient follow-up records were reviewed, and survival and hospitalization data were obtained from hospital records. The follow-up period was defined from the date of echocardiographic diagnosis to the last point of contact. Clinical outcomes included all-cause mortality and heart failure-related hospitalization. Progression to severe aortic stenosis and aortic valve replacement during follow-up were also recorded. Statistical analyses included t-tests, multivariable Cox regression analysis, and Kaplan-Meier survival analysis.

**Results:** The study included 177 patients with moderate aortic stenosis. Over a median follow-up of 4.7 years (interquartile range: 1.2-7.1 years), 11 patients (6.2%) died and 47 patients (26.6%) were hospitalized for heart failure. During follow-up, 60 patients (33.9%) progressed to severe aortic stenosis, and 40 patients (22.6%) underwent aortic valve replacement. Patients who experienced the composite outcome of all-cause mortality or heart failure hospitalization had significantly lower left atrial reservoir strain (25.1±4.2% *vs.* 31.2±6.2%), conduit strain (14.7±4.7% *vs.* 17.5±5.7%), and contraction strain (12.5±3.3% *vs.* 14.2±4.4%) than those who remained event-free (all p<0.05). In multivariable Cox regression analysis, age, left ventricular global longitudinal strain, and left atrial reservoir strain were independently associated with the composite outcome. Using a left atrial reservoir strain cut-off of 27%, patients with values <27% had an approximately 2.1-fold higher risk of the composite outcome (HR 2.099, 95% CI 1.110-3.990). Kaplan-Meier analysis demonstrated significantly worse event-free survival in patients with left atrial reservoir strain <27% (log-rank p<0.001).

**Conclusion:** Left atrial strain provides valuable prognostic information in patients with moderate aortic stenosis. Impaired left atrial reservoir strain was independently associated with mortality and heart failure hospitalization, suggesting that left atrial deformation may identify higher-risk patients even before progression to severe aortic stenosis. Assessment of left atrial strain parameters in addition to conventional echocardiographic parameters may improve risk stratification and help guide closer clinical and echocardiographic surveillance in patients with moderate aortic stenosis.

**Keywords:** Aortic stenosis, left atrial strain, speckle-tracking echocardiography.

## [OP-46]

***Best Oral Presentation Candidate***

### The association of ambulatory blood pressure pattern and HFA-PEFF score in diabetic patients

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**Background:** Type 2 diabetes (DM) is an established risk factor for heart failure with preserved ejection fraction (HFpEF). However, it generally remains under-recognised in this population. The "HFA-PEFF score" proposed by the Heart Failure Association (HFA) of the ESC facilitates objective diagnosis of HFpEF. Ambulatory blood pressure monitoring (ABPM) is an available tool in the management of hypertension, but its potential value in identifying patients with HFpEF has not been established. We sought to analyse the association of ABPM patterns with HFA-PEFF scores in patients with DM.

**Methods:** We prospectively and consecutively enrolled 123 patients with a new diagnosis of type 2 diabetes and left ventricular ejection fraction ≥50%. All underwent 24-hour ABPM, comprehensive echocardiography and NT-proBNP measurement. Non-dipping was defined as a nocturnal systolic BP fall <10%. HFA-PEFF scores (functional, morphological and biomarker

parameters, total: 0-6) were calculated according to the HFA/ESC consensus-algorithm (low: 0-1, intermediate: 2-4, high: 5-6 and diagnostic for HFpEF). Patients with non-Sinus rhythms, structural heart disease, renal failure and recent coronary syndrome (<6 months) were excluded. Continuous variables were compared with the Mann-Whitney U test and categorical variables with the chi-square test. Ordinal logistic regression was used to adjust for age, sex, body mass index, HbA1c, diabetes duration, 24-hour systolic BP and number of antihypertensive agents.

**Results:** Of 123 patients (58.6±8.7 years, female n=86, 70%), seventy-seven patients (62.6%) were non-dippers, including 19 reverse dippers. Daytime systolic BP was identical in dippers and non-dippers (131.7±13.8 *vs.* 132.7±17.4 mmHg, p=0.72). Non-dippers had significantly higher total HFA-PEFF scores (median 3 [3-4] *vs.* 2 [1-3], p<0.001), and each three score-groups contributed significantly (functional p=0.039, morphological p<0.001, biomarker p=0.001). The distribution across risk categories also differed significantly (p=0.005): a high, diagnostic score was six-fold more frequent among non-dippers (13.0% *vs.* 2.2%, p=0.052), while a low score was three-fold more frequent among dippers (28.3% *vs.* 9.1%). HFA-PEFF scores inclined progressively across dippers, non-dippers and reverse dippers (mean 2.07, 3.14 and 3.26; p<0.001). Non-dippers also had a larger left atrial volume index and higher NT-proBNP (89.4 [51.8-135.0] *vs.* 33.3 [18.2-51.0] pg/mL, both p<0.001). There was an inverse correlation with the magnitude of nocturnal systolic fall and the score (Spearman rho=-0.35, p<0.001). Non-dipping patterns remained independently associated with a higher HFA-PEFF score (odds ratio 6.46, 95% CI 2.85-14.63, p<0.001) after adjustment, and every 1% greater nocturnal fall reduced the odds of a higher score by 7% (odds ratio 0.93, 95% CI 0.88-0.98).

**Conclusion:** In patients with DM, a non-dipping BP pattern is found to be independently associated with a strikingly higher HFA-PEFF score despite an identical daytime BP, indicating a probable association of circadian pattern and diastolic remodelling. ABPM may serve as an accessible tool to select patients in need of a dedicated guideline-directed HFpEF diagnostic and therapeutic work-up.

**Keywords:** Heart failure, diabetes mellitus, NT-proBNP, ambulatory blood pressure monitoring, non-dipper hypertension.

## [OP-47]

***Best Oral Presentation Candidate***

### Cardiorenal-hepatic risk phenotypes and one-year outcomes following acute heart failure hospitalization

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**Background:** Left ventricular ejection fraction (LVEF) does not fully capture the burden of systemic congestion or prognosis in patients with acute heart failure (AHF). Concurrent renal and hepatic dysfunction has been proposed to identify a high-risk phenotype of systemic congestion that is independent of LVEF. In this study, we investigated the association between cardiorenal-hepatic risk phenotypes—based on renal (eGFR) and hepatic (FIB-4) dysfunction—and one-year mortality and composite outcome in patients hospitalized for AHF, independent of LVEF and age.

**Methods:** In a retrospective cohort of 359 patients hospitalized for AHF (mean age 72±10 years, 54% male), admission eGFR (CKD-EPI) and the FIB-4 index [(age×AST)/(platelets×√ALT)] were calculated. Patients were stratified into four phenotypes based on eGFR<60 mL/min/1.73 m$^2$ (renal dysfunction) and FIB-4 ≥2.67 (high probability of advanced hepatic fibrosis; standard cut-off in the literature): low organ-risk, predominantly renal, predominantly hepatic, and combined cardiorenal-hepatic. The primary outcome was one-year all-cause mortality; the secondary outcome was a composite of mortality and HF-related rehospitalization. Between-group comparisons were performed using the chi-square test, and the independent association of the combined phenotype versus the low-risk group was assessed using logistic regression adjusted for age and LVEF.

**Results:** Of the patients, 35% (n=127) were classified as low organ-risk, 44% (n=159) as predominantly renal, 8% (n=29) as predominantly hepatic, and 12% (n=44) as combined phenotype. One-year mortality was highest in the combined group (59%) and lowest in the low-risk group (28%), with intermediate rates in the predominantly renal and predominantly hepatic groups (45% and 41%, respectively) (p=0.001 across four groups). The combined phenotype was associated with a 3.65-fold increased risk of one-year mortality compared with the low-risk group (OR 3.65, 95% CI 1.79-7.46, p<0.001), and this association remained significant after adjustment for age and LVEF (adjusted OR 3.17, p=0.004). Composite outcome rates also differed significantly across phenotypes (range 50-68%, p=0.02; unadjusted OR 2.11 for combined vs. low-risk, 95% CI 1.02-4.35, p=0.04), but this association lost significance after adjustment for age and LVEF (p=0.18). In-hospital mortality was numerically highest in the combined group (14% *vs.* 2% in the low-risk group). LVEF was similar across the four groups (mean 39-47%, p=0.08), supporting that the phenotype carries prognostic information independent of LVEF.

**Conclusion:** Concurrent renal and hepatic dysfunction identifies a high-risk systemic congestion phenotype in AHF patients that is not captured by LVEF alone and is strongly and independently associated with one-year mortality, irrespective of LVEF and age. Combined assessment of eGFR and FIB-4 may provide a simple, cost-free risk stratification tool derived from routine laboratory data, potentially informing the intensity of post-discharge follow-up.

**Keywords:** Acute heart failure, renal failure, hepatic dysfunction, mortality.

## [OP-48]

***Best Oral Presentation Candidate***

### Prognostic value of parathyroid hormone in heart failure with reduced ejection fraction

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**Background:** To evaluate the association of parathyroid hormone (PTH) with long-term all-cause mortality in heart failure with reduced ejection fraction (HFrEF), its results in different age and renal function subgroups, and its incremental prognostic contribution beyond NT-proBNP and conventional clinical variables.

**Methods:** In this single-center retrospective cohort, 2,185 patients who presented to the heart failure outpatient clinic between January 2014 and February 2026 were screened, and 1,594 patients with a left ventricular ejection fraction ≤40% were included in the analysis. The primary endpoint was all-cause mortality. PTH and NT-proBNP levels were evaluated after natural logarithmic transformation in a Cox regression model adjusted for prespecified clinical variables. The incremental contribution of PTH was examined using the likelihood ratio test between nested models, and using net reclassification improvement (NRI) and integrated discrimination improvement (IDI) at 36 months.

**Results:** During a median follow-up of 36 months, 525 deaths occurred. Survival was lower in the high-PTH group (>82 pg/mL). In multivariable analysis, a one-unit increase in lnPTH was independently associated with mortality (hazard ratio [HR] 1.201; 95% confidence interval [CI] 1.007-1.432; p=0.0412); lnNT-proBNP was also an independent predictor (HR 1.360; 95% CI 1.249-1.482; p<0.0001). Adding PTH improved model fit ($\chi^2$=4.2419; p=0.0394). In contrast, the increases in reclassification and discrimination measures were not significant (NRI 0.0448; p=0.4006; IDI 0.00107; p=0.2296). The association between PTH and mortality was more pronounced in patients younger than 65 years (HR 1.402; 95% CI 1.088-1.806) and in those with an

estimated glomerular filtration rate ≥60 mL/min/1.73 m² (HR 1.454; 95% CI 1.129-1.873); the interaction p values for age and renal function were 0.026 and 0.038, respectively.

**Conclusion:** In patients with HFrEF, PTH was independently associated with long-term all-cause mortality and provided a limited additional contribution to the overall fit of the model containing NT-proBNP. Because a significant improvement in reclassification could not be demonstrated, external and prospective validation is required before routine clinical use.

**Keywords:** Heart failure, parathyroid hormone, NT-proBNP, mortality, prognosis.

## [OP-49]

### Modified Gustave Roussy immune score predicts in-hospital mortality in acute heart failure with reduced ejection fraction

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**Background:** Acute decompensated heart failure (ADHF) is associated with substantial in-hospital mortality. The modified Gustave Roussy immune score (m-GRIS), derived from albumin, lactate dehydrogenase (LDH) and the neutrophil-to-lymphocyte ratio (NLR), integrates inflammation, nutritional status and tissue injury; its prognostic value in ADHF has not been well defined.

**Methods:** This single-center retrospective cohort included 1,165 consecutive adults hospitalized with ADHF and left ventricular ejection fraction <40% (January 2023-November 2025). m-GRIS was calculated from admission laboratories within 24 h (1 point each for albumin <3.5 g/dL, LDH ≥250 U/L and NLR ≥2.7; total 0-3). The primary outcome was in-hospital all-cause mortality. Associations were assessed using cox regression with multivariable adjustment for clinically relevant covariates. Discrimination was evaluated using receiver operating characteristic (ROC) analysis and survival using Kaplan-Meier analysis (m-GRIS =0 *vs.* ≥1).

**Results:** In-hospital mortality occurred in 130 patients (11.2%). Non-survivors more frequently had m-GRIS ≥1 than survivors (85.4% *vs.* 55.7%, p<0.001). Each 1-point increase in m-GRIS was associated with mortality in univariable analysis (HR 2.456, 95% CI 2.027-2.977; p<0.001) and remained independently associated after adjustment (adjusted HR 1.898, 95% CI 1.418-2.540; p<0.001). m-GRIS showed fair discrimination (AUC 0.721, 95% CI 0.674-0.768; p<0.001). Kaplan-Meier analysis showed lower survival in patients with m-GRIS ≥1 (log-rank p<0.001).

**Conclusion:** m-GRIS independently predicts in-hospital mortality in ADHF with HFrEF and may support early bedside risk stratification.

**Keywords:** Acute decompensated heart failure, HFrEF, in-hospital mortality, inflammation, m-GRIS, neutrophil-to-lymphocyte ratio.

## [OP-50]

### Ten-year outcomes after pediatric heart transplantation with and without durable LVAD bridging

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**Background:** Durable left ventricular assist devices (LVADs) are increasingly used as a bridge to transplantation in children with end-stage heart failure. This study compared early and long-term outcomes after pediatric heart transplantation according to pretransplant LVAD support.

**Methods:** We retrospectively reviewed all patients aged <18 years who underwent orthotopic heart transplantation at our institution between January 2004 and December 2024. Of 34 recipients, 17 were bridged with a durable LVAD and 17 underwent transplantation without mechanical circulatory support. Perioperative characteristics, postoperative complications, mortality, overall survival, cardiac allograft vasculopathy (CAV), and treated rejection were compared between groups.

**Results:** LVAD-bridged recipients had more advanced pretransplant ventricular dysfunction and required longer cardiopulmonary bypass and aortic cross-clamp times. Blood transfusions were also higher in the LVAD group (median, 2 [IQR, 1-3] *vs.* 1 [IQR, 0-2] units; p=0.030). Nevertheless, early mortality, major postoperative complications, and intensive care unit and hospital lengths of stay were comparable between groups. Ten-year survival was 70.6% among LVAD-bridged recipients and 82.4% among recipients transplanted without LVAD support, with no statistically significant difference (log-rank p=0.365). Long-term freedom from CAV and treated rejection was also comparable.

**Conclusion:** Durable LVAD support enabled successful transplantation in higher-risk pediatric candidates without significantly compromising early or long-term post-transplant outcomes. These findings support LVAD bridging as a safe and effective strategy for children awaiting heart transplantation.

**Keywords:** Pediatric heart transplantation, LVAD, ventricular assist device, heart failure.

## [OP-51]

### Association of preimplantation obesity with mortality and adverse events after HeartMate 3 implantation

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**Background:** The prognostic effect of obesity during contemporary durable mechanical circulatory support remains incompletely defined. This study investigated whether preimplantation obesity influences mortality and adverse events after HeartMate 3 (HM3) implantation.

**Methods:** We retrospectively evaluated 215 adults who underwent primary HM3 implantation between January 2016 and October 2025. Patients were stratified as normal/underweight (BMI <25 kg/m²; n=94), overweight (BMI 25.0-29.9 kg/m²; n=81), or obese (BMI ≥30 kg/m²; n=40). The primary endpoint was on-device mortality, with heart transplantation considered a competing event. Cumulative incidence functions and adjusted cause-specific hazard models were used for survival analyses. Secondary endpoints comprised early postoperative complications and major device-related adverse events.

**Results:** Median duration of support was 603 days (interquartile range, 188-1,210). On-device mortality progressively increased across BMI categories (20.2%, 27.2%, and 42.5%, respectively). Thirty-day mortality was highest among patients with obesity (13.8%, 6.2%, and 22.5%; p=0.030), as was prolonged mechanical ventilation (13.8%, 3.7%, and 20.0%; p=0.009). Five-year cumulative mortality was 24.7%, 41.9%, and 48.2% across BMI groups, respectively. Obesity was independently associated with a higher average cause-specific hazard of on-device death compared with BMI <30 kg/m$^2$ (adjusted hazard ratio, 1.81; 95% confidence interval, 1.01-3.24; p=0.046). Driveline infection and most device-related adverse events were comparable across groups. Ischemic stroke was more frequent in obesity, although event numbers were limited.

**Conclusion:** Obesity was associated with greater early postoperative vulnerability and increased adjusted mortality during HM3 support. These findings support incorporating BMI into comprehensive preimplantation risk assessment without considering obesity an isolated contraindication.

**Keywords:** Obesity, BMI, heart failure, ventricular assist device.

## [OP-52]

### Left atrial reservoir strain predicts new-onset atrial fibrillation in hemodialysis patients: A 10-year follow-up study

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**Background:** Atrial fibrillation (AF) is common in patients undergoing hemodialysis and is associated with adverse clinical outcomes. Left atrial reservoir strain (LASr) is a sensitive marker of left atrial mechanical dysfunction and may identify susceptibility to AF before the development of clinically apparent atrial arrhythmia. To investigate whether impaired LASr predicts new-onset AF during long-term follow-up in patients undergoing chronic hemodialysis who were in sinus rhythm at baseline.

**Methods:** This longitudinal study included 85 patients undergoing chronic hemodialysis, all of whom were in sinus rhythm at baseline. Comprehensive transthoracic echocardiography and two-dimensional speckle-tracking analysis were performed at study entry. Patients were categorized according to LASr using a cut-off value of 28%, as reduced LASr (<28%) or preserved LASr (≥28%). The primary outcome was new-onset AF during approximately 10 years of follow-up. AF-free survival was assessed using Kaplan-Meier analysis and compared with the log-rank test. Cox proportional hazards regression was used to evaluate the independent association between reduced LASr and incident AF after adjustment for age, sex, left ventricular ejection fraction (LVEF), left ventricular global longitudinal strain (LVGLS), left atrial volume index (LAVI), and left atrial major diameter.

**Results:** During follow-up, new-onset AF occurred in 29 of 80 patients (36.3%). AF developed in 22 of 47 patients (46.8%) with reduced LASr compared with 7 of 33 patients (21.2%) with preserved LASr. Kaplan-Meier analysis demonstrated significantly lower AF-free survival in patients with reduced LASr (log-rank $\chi^2$=7.824, p=0.005). In multivariable Cox proportional hazards analysis, reduced LASr remained independently associated with new-onset AF after adjustment for age, sex, LVEF, LVGLS, LAVI, and left atrial major diameter (adjusted HR 2.57, 95% CI 1.04-6.35; p=0.041). None of the other variables included in the multivariable model were independently associated with incident AF.

**Conclusion:** In chronic hemodialysis patients who were in sinus rhythm at baseline, reduced LASr was associated with a significantly increased risk of new-onset AF during 10-year follow-up. An LASr <28% identified patients with an approximately 2.6-fold higher adjusted risk of incident AF, independently of conventional measures of left ventricular and left atrial structure and function. These findings suggest that LASr may be useful for long-term AF risk stratification in the hemodialysis population.

**Keywords:** Left atrial reservoir strain (LASr), atrial fibrillation, hemodialysis, speckle-tracking echocardiography, left atrial dysfunction, risk stratification.

## [OP-53]

### Prevalence, clinical profile, and determinants of chronic cardiorenal syndrome in patients with heart failure

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**Background:** Cardiorenal syndrome (CRS) is a common and clinically meaningful complication in patients with heart failure (HF), contributing to increased morbidity, healthcare utilization, and poor outcomes. Despite its clinical relevance, real-world data describing the prevalence and clinical correlates of chronic CRS remain limited. To determine the prevalence of chronic CRS and to characterize its associated clinical, functional, and comorbidity profiles in a contemporary HF population.

**Methods:** This cross-sectional study analyzed 2,259 patients with chronic HF and available CRS status enrolled in a multicenter registry. Renal dysfunction was defined as an estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m$^2$, consistent with chronic CRS phenotypes. Demographic characteristics, HF phenotype, NYHA functional class, comorbidities, prior HF-related hospitalization, emergency department (ED) admissions, and treatment-related variables were compared between patients with and without CRS using chi-square analyses.

**Results:** Chronic CRS was present in 851 of 2,259 patients (37.7%). Patients with CRS had a substantially greater clinical and functional burden. They were more frequently female (46.7% *vs.* 31.4%, p<0.001) and had a marked shift toward advanced NYHA functional classes (p<0.001), with NYHA III-IV symptoms occurring more frequently and NYHA I being considerably less common than in patients without CRS. Healthcare utilization was significantly greater in patients with CRS. Among patients with available hospitalization data, HF-related hospitalization was more frequent in the CRS group (46.0% *vs.* 31.2%, p<0.001), while ED admission occurred in 52.6% *vs.* 33.5% (p<0.001). Oral diuretic dose escalation

was also more frequent in patients with CRS (30.2% *vs.* 16.9%, p<0.001). CRS was associated with a higher prevalence of hypertension (74.9% *vs.* 50.5%, p<0.001), diabetes mellitus (47.5% *vs.* 33.3%, p<0.001), coronary artery disease (63.8% *vs.* 54.9%, p<0.001), atrial fibrillation (45.6% *vs.* 26.7%, p<0.001), anemia (47.0% *vs.* 22.3%, p<0.001), iron deficiency (35.3% *vs.* 28.3%, p=0.001), rheumatic/degenerative valvular disease (22.4% *vs.* 11.6%, p<0.001), malignancy (8.0% *vs.* 4.8%, p=0.002), and thyroid disease (13.6% *vs.* 9.4%, p=0.002). A history of cardiac surgery was also more frequent among patients with CRS (27.5% *vs.* 19.8%, p<0.001). The prevalence of CRS differed significantly across HF phenotypes (p<0.001). CRS was most prevalent in HFpEF (51.6%; 292/566), compared with HFmrEF (33.5%; 76/227) and HFrEF (32.9%; 483/1,466).

**Conclusion:** Chronic cardiorenal syndrome affects more than one-third of patients with heart failure and is associated with a distinct high-risk clinical phenotype characterized by greater functional impairment, higher healthcare utilization, and a substantially greater burden of cardiovascular and non-cardiovascular comorbidities. CRS was particularly prevalent among patients with HFpEF. These findings underscore the importance of early recognition of renal involvement and support integrated heart–kidney management strategies in patients with HF.

**Keywords:** Heart failure, chronic cardiorenal syndrome, renal dysfunction, HFpEF, HFrEF, comorbidities, healthcare utilization, clinical phenotype.

## [OP-54]

### Short-term prognostic value of serum chloride compared with serum sodium in patients with worsening heart failure

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**Background:** Hyponatremia is a well-established marker of adverse prognosis in worsening heart failure (WHF). Serum chloride has recently emerged as a potential prognostic biomarker reflecting neurohormonal activation and diuretic responsiveness. We aimed to compare the prognostic performance of admission serum sodium and chloride levels for predicting 120-day clinical outcomes in patients hospitalized with WHF.

**Methods:** This retrospective cohort study included 136 consecutive patients hospitalized for WHF at a tertiary referral center. Patients were stratified according to admission serum sodium (<135 *vs.* ≥135 mmol/L) and serum chloride (<96 *vs.* ≥96 mmol/L). The primary endpoint was a composite of 120-day adverse heart failure outcomes, including all-cause mortality, heart transplantation, left ventricular assist device implantation, or heart failure rehospitalization/emergency department admission. Logistic regression and Cox proportional hazards analyses were performed to evaluate prognostic associations. Sodium- and chloride-based models were compared using the Akaike information criterion (AIC) and Harrell's C-index.

**Results:** The median age was 55 (42.8-65.0) years, 80.9% were male, and the median left ventricular ejection fraction was 18%; 97.8% had heart failure with reduced ejection fraction. Hyponatremic patients had longer hospital stays (15 *vs.* 7 days, p<0.001), higher inotrope requirements (63.8% *vs.* 12.8%, p<0.001), higher rates of the primary endpoint (53.4% *vs.* 16.7%, p<0.001), and higher mortality (22.4% *vs.* 5.1%, p=0.003). Hypochloremic patients had longer hospital stays (17 *vs.* 7 days, p<0.001) and higher mortality (32.3% *vs.* 6.7%, p<0.001). As a continuous variable, each 1 mmol/L increase in serum chloride was independently associated with lower odds of the primary composite endpoint (OR 0.87, 95% CI 0.80-0.94; p<0.001). In the adjusted Cox model, serum chloride remained independently associated with the primary composite endpoint (HR 0.90, 95% CI 0.85-0.95; p<0.001). The chloride-based model showed better prognostic performance than the sodium-based model (AIC: 399.5 *vs.* 405.5; C-index: 0.709 *vs.* 0.694). When both electrolytes were included in the same model, serum chloride remained independently associated with the primary composite endpoint (p=0.044), whereas serum sodium lost statistical significance.

**Discussion:** In this cohort of predominantly advanced heart failure with reduced ejection fraction receiving high rates of guideline-directed medical therapy, serum chloride provided prognostic information distinct from, and potentially superior to, serum sodium. Although hypochloremia defined by a categorical threshold was not significantly associated with the composite endpoint, the significant association observed when chloride was analyzed as a continuous variable suggests loss of prognostic information due to dichotomization.

**Conclusion:** Admission serum chloride was independently associated with adverse 120-day clinical outcomes in patients hospitalized for worsening heart failure and demonstrated superior prognostic model performance compared with serum sodium. As a routinely measured and cost-neutral laboratory parameter, serum chloride may contribute to early risk stratification. However, its incremental prognostic value in clinical risk assessment should be confirmed in larger prospective cohorts.

**Keywords:** Chlorides, heart failure, mortality, prognosis.

## [OP-55]

### Prognostic impact of acute heart failure on mortality in patients with acute coronary syndrome: A multivariable survival analysis

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**Background:** Acute coronary syndrome remains a leading cause of cardiovascular mortality worldwide. This study aims to investigate the independent prognostic impact of acute heart failure on in-hospital mortality among acute coronary syndrome patients, utilizing a multivariable Cox proportional hazards regression model.

**Methods:** A retrospective analysis was conducted on a cohort of 381 acute coronary syndrome patients. Demographic characteristics, clinical parameters, and laboratory findings were evaluated. Univariate and multivariable Cox proportional hazards regression analyses were performed to identify independent predictors of mortality. Kaplan-Meier survival curves with the log-rank test were used to illustrate survival probabilities stratified by acute heart failure status.

**Results:** During the hospital stay, patients presenting with acute heart failure exhibited a significantly reduced survival probability compared to those without acute heart failure (p<0.001). In the multivariable Cox regression model (adjusted for age, sex, acute coronary syndrome subtype, and creatinine levels), acute heart failure emerged as an independent predictor, increasing the risk of mortality by 4.4-fold (aHR=4.40, 95% CI: 1.66-11.7, p=0.003). Furthermore, ST-segment elevation myocardial infarction (aHR=7.38, 95% CI: 2.37-23.0, p=0.001) and elevated creatinine levels (aHR=1.97, 95% CI: 1.28-3.03, p=0.002) were identified as other significant independent risk factors for mortality.

**Conclusion:** Acute heart failure is an independent predictor of in-hospital mortality in patients with acute coronary syndrome. These findings highlight the importance of early identification of acute heart failure and comprehensive risk assessment in patients with acute coronary syndrome.

**Keywords:** Acute coronary syndrome, acute heart failure, in-hospital mortality, survival analysis.

## [OP-56]

### Influenza vaccination in heart failure after the COVID-19: Associations with pandemic experience and behavioral changes

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**Background:** Influenza is associated with increased morbidity and cardiovascular events in patients with heart failure (HF), and annual influenza vaccination is recommended for this high-risk population. The COVID-19 pandemic substantially altered perceptions of infectious diseases and preventive health behaviors. We aimed to determine influenza vaccination uptake among patients with HF assessed in the post-pandemic period and to investigate the associations of COVID-19 experience and retrospectively reported pandemic-related behavioral changes with influenza vaccination status.

**Methods:** This cross-sectional study, conducted in the post-pandemic period, included 548 patients with HF. Participants completed a structured questionnaire assessing demographic and clinical characteristics, history of COVID-19 infection, COVID-19 infection among close contacts, and retrospectively reported concerns and behavioral changes during the pandemic. Awareness and perceived safety of influenza vaccination, influenza vaccination during the preceding year, and reasons for non-vaccination were also assessed.

**Results:** The mean age was 68.2±9.7 years, and 49.5% of patients were male. Hypertension, diabetes mellitus, and hyperlipidemia were present in 63.7%, 34.7%, and 41.4% of patients, respectively. Regular physician follow-up was reported by 87.0% of participants. Previous COVID-19 infection was reported by 32.7%, while 46.9% reported COVID-19 infection among close contacts. Although 95.3% of participants were aware of influenza vaccination, only 48.4% (n=265) had received an influenza vaccine during the preceding year. Participants retrospectively reported various protective behavioral changes during the pandemic, including mask use, increased hand hygiene, and restriction of physical and social contact. Concerns regarding vaccine safety and effectiveness and the absence of physician recommendation were among the reported reasons for non-vaccination.

**Discussion and Conclusion:** In the post-pandemic period, influenza vaccination uptake remained below 50% among patients with HF despite high awareness. The findings highlight the relevance of examining potential associations between pandemic experiences, preventive behaviors adopted during the pandemic, and subsequent influenza vaccination practices. However, causal inferences cannot be made because of the cross-sectional design and retrospective assessment of pandemic-related behaviors. Strategies aimed at improving influenza vaccination uptake in patients with HF should address vaccine confidence and strengthen physician recommendation.

**Keywords:** Heart failure, influenza vaccination, COVID-19, vaccine hesitancy, health behavior.

## [OP-57]

### The relationship between pulse pressure level and mortality in patients with heart failure

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**Background:** The aim of this study was to investigate the association between pulse pressure and one-year mortality in patients with heart failure with reduced ejection fraction (HFrEF).

**Methods:** A total of 99 patients diagnosed with HFrEF were included in this retrospective study. Demographic, clinical, and laboratory characteristics were recorded. Patients were divided into two groups according to the presence or absence of mortality within one year following the index admission. Pulse pressure was calculated as the difference between systolic and diastolic blood pressure. Independent determinants of mortality were evaluated using logistic regression analysis. The predictive performance of pulse pressure for one-year mortality was assessed using receiver operating characteristic (ROC) curve analysis.

**Results:** Compared with survivors, patients who died had significantly higher levels of creatinine, high-sensitivity C-reactive protein (hs-CRP), B-type natriuretic peptide (BNP), total cholesterol, and low-density lipoprotein cholesterol (LDL-C), whereas sodium and pulse pressure levels were lower. In multivariable logistic regression analysis, higher hs-CRP, BNP, and LDL-C levels, as well as lower sodium and pulse pressure levels, were identified as independent predictors of one-year mortality. ROC curve analysis demonstrated that a pulse pressure cut-off value of 47.5 mmHg predicted one-year mortality, with an area under the curve (AUC) of 0.638 (95% confidence interval [CI]: 0.528-0.748; p=0.024).

**Discussion:** Low pulse pressure may reflect reduced cardiac output and diminished cardiac reserve and may therefore be associated with an adverse prognosis in patients with HFrEF. The 47.5 mmHg cut-off value identified in our study suggests that pulse pressure may serve as a simple, readily obtainable, and non-invasive prognostic marker. Furthermore, the association of elevated hs-CRP and BNP levels and reduced serum sodium levels with mortality supports the prognostic significance of inflammation and neurohormonal activation in HFrEF.

**Conclusion:** Low pulse pressure was identified as an independent predictor of one-year mortality in patients with HFrEF. Pulse pressure may represent a simple, readily available, and non-invasive prognostic parameter for risk stratification in this patient population. Further studies involving larger cohorts and longer follow-up periods are warranted to validate these findings.

**Keywords:** Heart failure with reduced ejection fraction, pulse pressure, one-year mortality.