



**Day 1
UPDATES**

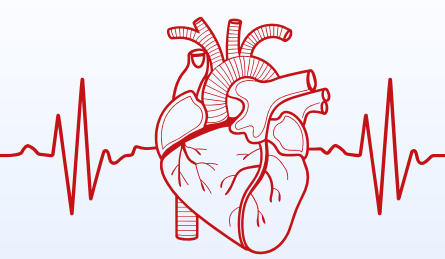




**Munich,
Germany**



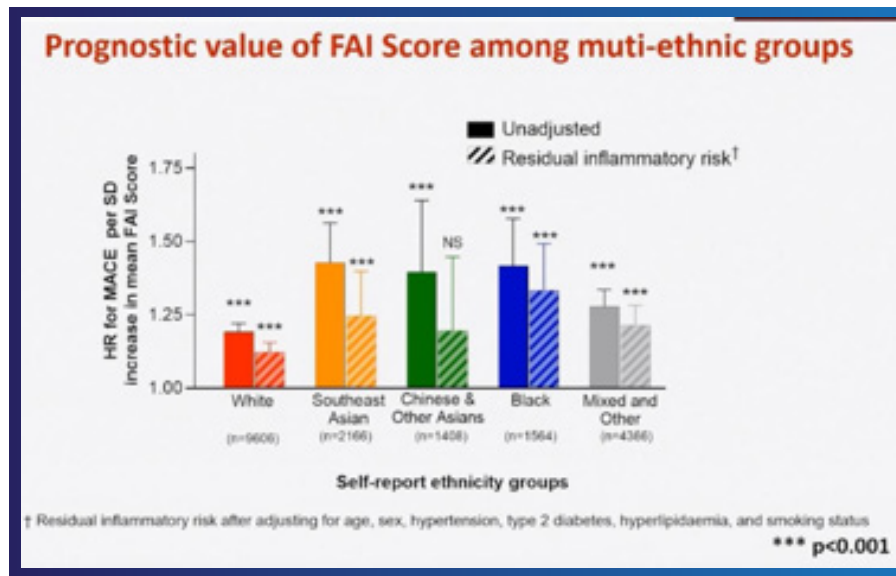
**28TH - 31ST
Aug, 2026**



ESC 2026

EUROPEAN SOCIETY OF CARDIOLOGY

Our Global Marketing Arms



Cardiovascular risk assessment based on coronary artery calcification may not fully capture atherosclerotic risk across ethnic groups, particularly in individuals without coronary calcification. Coronary CT angiography (CCTA) enables assessment of coronary plaque burden, stenosis severity and high-risk plaque characteristics, while artificial intelligence (AI)-based analysis can quantify non-calcified plaque and provide additional information for risk stratification. This study evaluated the potential of AI-enabled CCTA interpretation and perivascular fat attenuation index (FAI) assessment to improve cardiovascular risk stratification across ethnic groups and identify residual inflammatory risk. The findings were presented at ESC Congress 2026, held in Munich, Germany, from August 28–31, 2026.

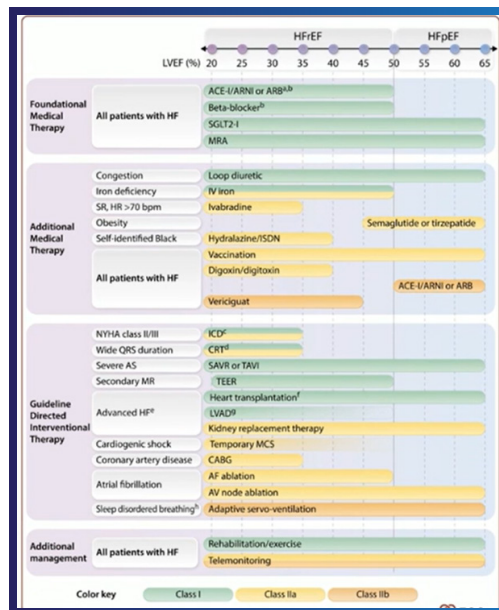
Evidence from multi-ethnic cardiovascular studies and the ORFAN cohort was evaluated. The ORFAN cohort included 250,000 patients with CCTA linked to 15-year outcomes data, comprising approximately 200,000 patients from the UK and 50,000 from international sites. Validation of a new CCTA-based cardiovascular risk prediction model against 10-year outcomes was completed in the first 100,000 UK and 30,000 international patients. AI-enabled CCTA analysis assessed plaque burden, stenosis severity, high-risk plaque characteristics and non-calcified plaque, while FAI was used to assess vascular inflammation.

Among 31,781 individuals investigated in the South Asian community, the overall prevalence of ASCVD and premature ASCVD was 7.1% and 2.5%, respectively. Hyperlipidaemia was reported in 13,677 (43.0%), hypertension in 7,056 (22.2%), diabetes mellitus in 4,934 (15.5%), and family history of cardiovascular disease in 3,350 (10.5%). Prevalence of ASCVD was 3,350 (10.5%), while premature ASCVD was 802 (2.52%). CCTA stenosis was categorised as minimal (1–24%), mild (25–49%), moderate (50–69%) or severe ($\geq 70\%$). Increasing mean FAI score was associated with higher MACE risk across ethnic groups, including White (n=9606), Southeast Asian (n=2166), Chinese and Other Asian (n=1408), Black (n=1564) and Mixed and Other (n=4366) populations ($p<0.001$). This association remained after adjustment for age, sex, hypertension, type 2 diabetes, hyperlipidaemia and smoking status.

Conclusion

The findings suggest that AI-enabled CCTA analysis can facilitate quantification of non-calcified plaque and may improve cardiovascular risk stratification, particularly among non-white populations without coronary calcification. FAI provides additional insight into residual inflammatory risk and may support more personalised preventive management across diverse ethnic groups.

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The 2026 ESC Guidelines for the Management of Heart Failure introduce new concepts, classifications, nomenclature, and several important recommendations across the heart failure continuum. HFrEF has been expanded to include LVEF up to 50%, HFmrEF has been removed, and HFpEF includes LVEF $\geq 50\%$. Decompensated heart failure replaces acute heart failure, while stages A–D emphasize prevention and timely management.

For prevention, strict blood pressure control with a systolic blood pressure target <130 mmHg is recommended in patients with hypertension or stage B HF (Class I, Level A). An SGLT2 inhibitor should be considered in patients with T2DM and at least one other cardiovascular risk factor to reduce the risk of HF or CV death (Class IIa, Level A). ACE inhibitors/ARBs and beta-blockers are recommended in stage B HF with LVEF $\leq 40\%$ (Class I, Level A).

New treatment nomenclature introduces foundational medical therapy (FMT), additional medical therapy (AMT), and guideline-directed interventional therapy (GDIT). SGLT2 inhibitors and MRAs are recommended in symptomatic HF independent of LVEF to reduce HF hospitalization or CV death (Class I, Level A). Beta-blockers and ACE inhibitors/ARNI are recommended in symptomatic HFrEF (Class I, Level A).

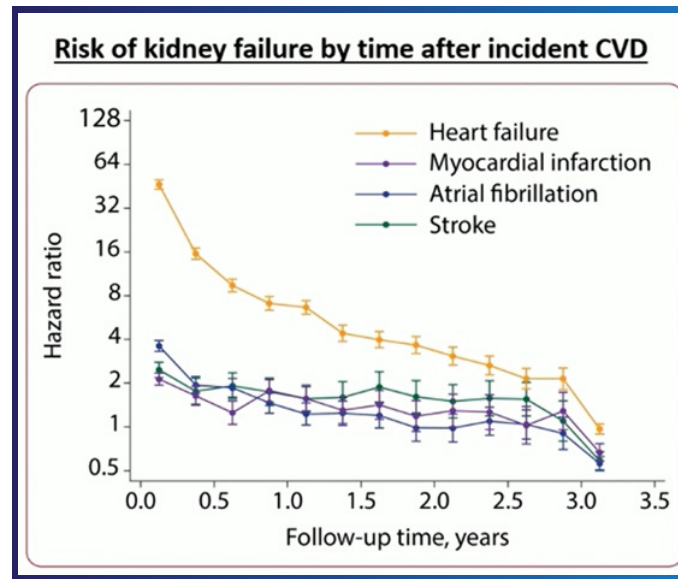
In decompensated HF, urinary sodium-guided diuretic therapy may be considered to improve natriuresis and diuresis (Class IIb, Level B1). Short-term intravenous acetazolamide or oral hydrochlorothiazide should be considered in patients with fluid overload previously treated with loop diuretics (Class IIa, Level B1). In-hospital initiation of SGLT2 inhibitors is recommended after initial stabilization (Class I, Level B1). A multidisciplinary Shock Team is recommended for potential temporary mechanical circulatory support candidates (Class I, Level C), while temporary microaxial flow pump support should be considered in selected patients with cardiogenic shock caused by STEMI (Class IIa, Level B1).

Semaglutide or tirzepatide should be considered in symptomatic HF with LVEF $\geq 45\%$ and BMI ≥ 30 kg/m², regardless of diabetes status (Class IIa, Level B1). Early consultation with an advanced HF centre is recommended in patients with advanced or at-risk HF (Class I, Level A). TTR silencers or stabilizers are recommended for transthyretin cardiac amyloidosis with NYHA class I–III (Class I, Level B).

Conclusion

The 2026 ESC HF Guidelines emphasize prevention and early, LVEF-independent treatment, with SGLT2 inhibitors and MRAs recommended in symptomatic HF (Class I, Level A). New recommendations include in-hospital SGLT2i initiation after stabilization, enhanced decongestion strategies, and semaglutide/tirzepatide for selected patients with HF and obesity.

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The 2026 ESC Guidelines for the Management of Cardiovascular Disease (CVD) and Chronic Kidney Disease (CKD), developed in collaboration with the European Renal Association, emphasize the need for integrated cardiorenal care. CKD is a major cardiovascular risk factor, increasing the burden of heart failure, left ventricular hypertrophy, atrial fibrillation, sudden cardiac death, and atherosclerotic CVD. Traditional cardiovascular risk assessment may underestimate risk in CKD, as eGFR and albuminuria are key predictors of cardiovascular and kidney outcomes.

With approximately 840 million people affected by CKD globally, the guideline highlights early detection and timely intervention. The “STAMP on CKD” framework provides a structured approach: Screen, Triage and stage, Address risk, Modify CVD management, and Plan health services.

All patients with CVD should undergo CKD screening using eGFR and urine albumin-to-creatinine ratio (uACR) (Class I) at diagnosis and annually. CKD is defined by eGFR <60 mL/min/1.73 m² or uACR ≥ 30 mg/g persisting for ≥ 3 months. Risk stratification should follow KDIGO classification based on eGFR and albuminuria.

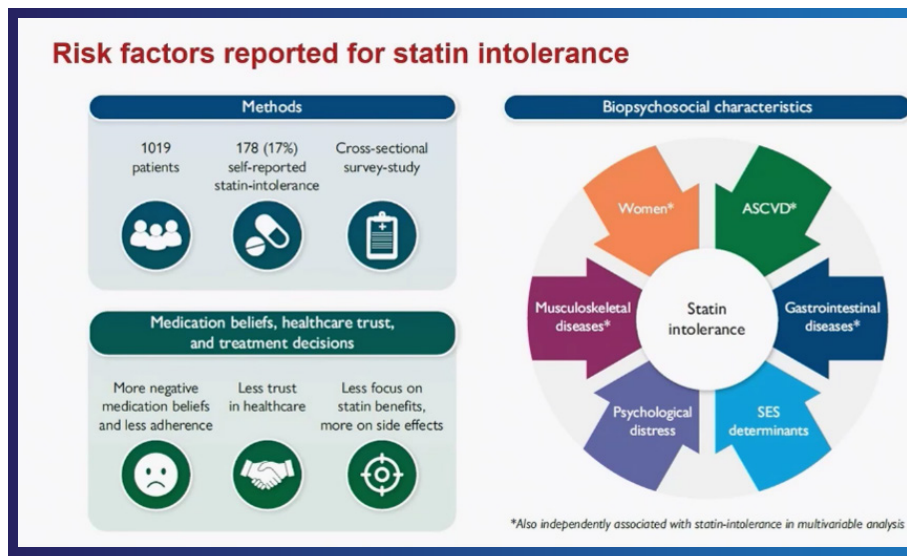
The guidelines recommend early use of evidence-based therapies, including SGLT2 inhibitors (eGFR ≥ 20 mL/min/1.73 m²), ACE inhibitors/ARBs, GLP-1 receptor agonists, and finerenone (nsMRA) in appropriate patients to reduce CKD progression and cardiovascular risk. Blood pressure target is $<130/80$ mmHg, with individualized HbA1c targets of 6.5–8.0%.

CVD management should be adapted according to kidney function, including optimized heart failure therapy, appropriate anticoagulation strategies, and modified antiplatelet approaches due to increased bleeding risk in CKD. In patients with eGFR <30 mL/min/1.73 m², low/iso-osmolar contrast strategies are recommended during cardiovascular procedures.

Conclusion

Overall, the guideline promotes early diagnosis, personalized treatment, multidisciplinary collaboration, and coordinated care to reduce cardiovascular events, kidney failure, and the growing global burden of cardiorenal disease.

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Statins are effective, safe, and inexpensive drugs for lowering cardiovascular disease risk, yet perceived statin intolerance may limit their use. This reviewed the definition, prevalence, risk factors, and practical management of statin intolerance, with emphasis on distinguishing true adverse effects from placebo-related symptoms. This session was presented by Prof Jeanine Roeters van Lennep at the European Society of Cardiology (ESC) Congress 2026 in Munich, Germany.

Statin intolerance was defined as adverse effects resolving or improving with dose reduction or discontinuation, classified as complete or partial. Diagnosis requires a trial of more than two statins, including one at the lowest dose. In 105,329 MI-hospitalized patients, intolerance was linked to 56% higher recurrent CHD events and 50% higher recurrent MI risk versus high adherence. Adverse effects included muscle symptoms (myalgia, myopathy, rhabdomyolysis), transaminase elevation, new-onset diabetes, alopecia, and weight gain. Observational prevalence was 17% (14–19%) across 64 cohorts, versus 4.9% (4.0–6.0%) across 112 randomized controlled trials. N-of-1 trials showed placebo effects, with 90% of SAMSON symptoms occurring similarly with statin and placebo. StatinWISE found 66% restarted or wanted to restart despite muscle-pain complaints. Across 176 studies (4,143,517 patients), prevalence was 9.1%, with female sex, high statin dose, hypothyroidism, and calcium-channel blockers as leading risk factors.

Management emphasized patient education, explaining cardiovascular risk and statin benefits and safety, addressing misinformation, and discussing the placebo effect. When symptoms occur, patients should be taken seriously while considering alternative causes, with options including a different statin or lower dose. For those unable or unwilling to take statins, non-statin therapies may be used according to risk category, distance to target, and local reimbursement criteria.

Conclusion

A practical approach to statin intolerance requires recognition of true adverse effects and the placebo effect, alongside patient education and individualized treatment strategies to support cardiovascular risk reduction.

Our Global Marketing Arms

2025 ESC/EAS Guidelines for the management of dyslipidaemias

Cardiovascular risk estimation: New recommendations

Recommendations	Class	Level
Recommendations for cardiovascular risk estimation in persons without known cardiovascular disease		
SCORE2 is recommended in apparently healthy people <70 years of age without established ASCVD, DM, CKD, genetic/rare lipid or BP disorders for estimation of 10-year fatal and non-fatal CVD risk.	I	B
SCORE2-OP is recommended in apparently healthy people ≥70 years of age without established ASCVD, DM, CKD, genetic/rare lipid or BP disorders for estimation of 10-year fatal and non-fatal CVD risk.	I	B

The 2025 Focused Update of the 2019 ESC/EAS Guidelines for the Management of Dyslipidemias introduces important changes to cardiovascular risk assessment and lipid management, with greater emphasis on individualized risk stratification, lipoprotein(a) [Lp(a)], and earlier intensification of lipid-lowering therapy. This update was presented at the European Society of Cardiology (ESC) Congress 2026, held from 28th to 31st August 2026 in Munich, Germany.

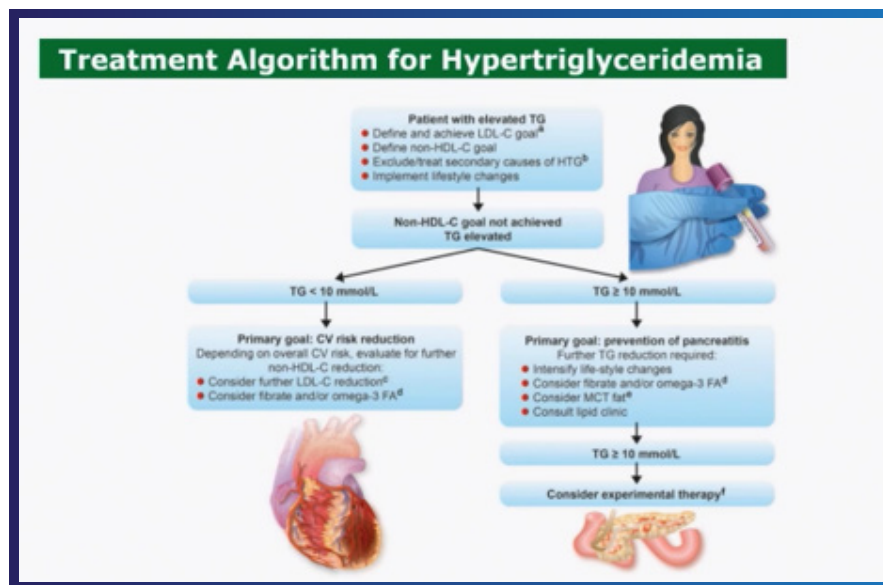
The updated recommendations refine cardiovascular risk estimation using SCORE2 and SCORE2-OP and further defines risk categories across primary prevention. Subclinical coronary atherosclerosis and coronary artery calcium may be considered as risk modifiers in selected individuals, while additional demographic, clinical, and biomarker-based risk modifiers can improve risk classification around treatment thresholds.

A key update is the recognition of Lp(a) levels >50 mg/dL (105 nmol/L) as a cardiovascular risk-enhancing factor in adults, with higher concentrations associated with progressively greater lifetime cardiovascular risk. The guideline also expands pharmacologic options, recommending bempedoic acid for patients unable to take statins and supporting its addition to maximally tolerated statin therapy, while evinacumab may be considered in selected patients with homozygous familial hypercholesterolemia. High-dose icosapent ethyl is recommended for selected high- or very-high-risk patients with elevated triglycerides despite statin therapy.

Conclusion

Overall, the 2025 update reinforces a more personalized, earlier, and intensive approach to dyslipidemia management, integrating lifetime risk, Lp(a), advanced risk modifiers, and expanded treatment options to improve LDL-C goal attainment and reduce residual cardiovascular risk. New recommendations in HIV and cardio-oncology further broaden the scope of preventive lipid management.

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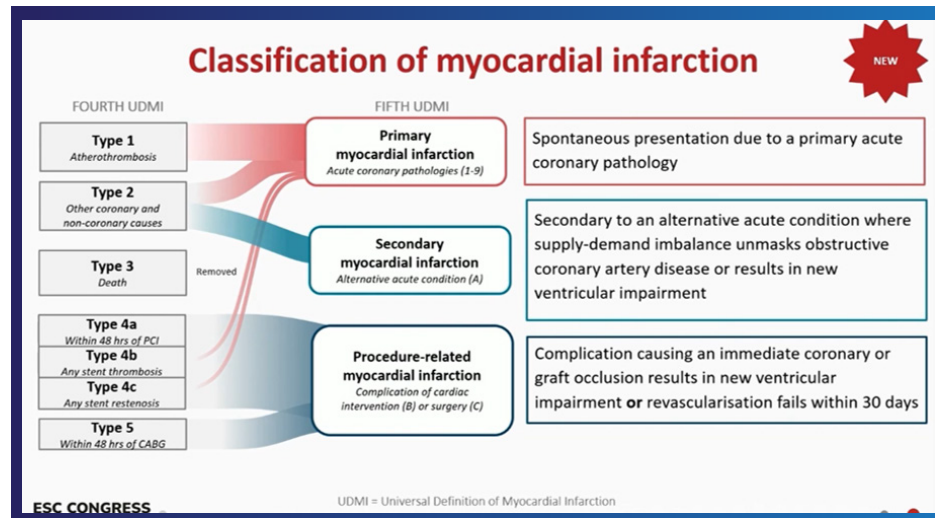
Hypertriglyceridemia is a common condition and is associated with both atherosclerotic cardiovascular disease (ASCVD) and acute pancreatitis. Its management requires distinguishing cardiovascular risk from pancreatitis risk, as triglyceride-rich lipoproteins contribute to atherogenic risk primarily through their cholesterol and apolipoprotein content. This session was presented by Dr. K Parhofer at the European Society of Cardiology (ESC) Congress 2026, held from 28th to 31st August 2026 in Munich, Germany.

Management should begin with assessment of overall ASCVD risk, identification and treatment of secondary causes, lifestyle modification, and achievement of appropriate LDL-C and non-HDL-C targets. For patients with persistent hypertriglyceridemia, further triglyceride lowering may be considered with fibrates and/or high-dose omega-3 fatty acids, while treatment intensity should reflect the underlying cardiovascular and pancreatitis risk.

Triglyceride-rich lipoproteins were highlighted as important contributors to atherosclerotic risk, with non-HDL-C and apoB providing better risk prediction than triglyceride concentration alone. Omega-3 fatty acids produce dose-dependent triglyceride reductions, while cardiovascular outcome data have demonstrated benefits with EPA-based therapy but not consistently with EPA+DHA preparations. Emerging therapies targeting APOC3 and ANGPTL3 have demonstrated substantial reductions in triglycerides and atherogenic lipoproteins.

Conclusion

Effective hypertriglyceridemia management requires a risk-based strategy rather than focusing on triglyceride concentration alone. For ASCVD prevention, priority should be given to LDL-C, non-HDL-C, and apoB reduction, whereas severe hypertriglyceridemia requires additional triglyceride-lowering strategies to prevent pancreatitis. Emerging APOC3- and ANGPTL3-targeted therapies may further expand treatment options for patients with difficult-to-control disease.



The Fifth Universal Definition of Myocardial Infarction (UDMI) 2026, jointly developed by ESC/ACC/AHA/WHF, introduces a simplified clinical classification of myocardial infarction (MI), replacing the previous numerical classification. MI is now categorized as **primary, secondary, or procedure-related**, based on underlying pathophysiology and clinical setting.

Primary MI refers to spontaneous MI due to an acute coronary pathology, including atherothrombosis, spontaneous coronary artery dissection, coronary embolism, vasospasm, and late (>30 days) stent thrombosis, restenosis, or graft failure. Diagnosis requires **acute myocardial injury**, demonstrated by a rise and/or fall in cardiac troponin with ≥ 1 value above the sex-specific 99th-percentile upper reference limit, together with clinical or ECG evidence of myocardial ischaemia. Coronary angiography and/or cardiac imaging are encouraged to confirm the diagnosis and define the underlying pathology.

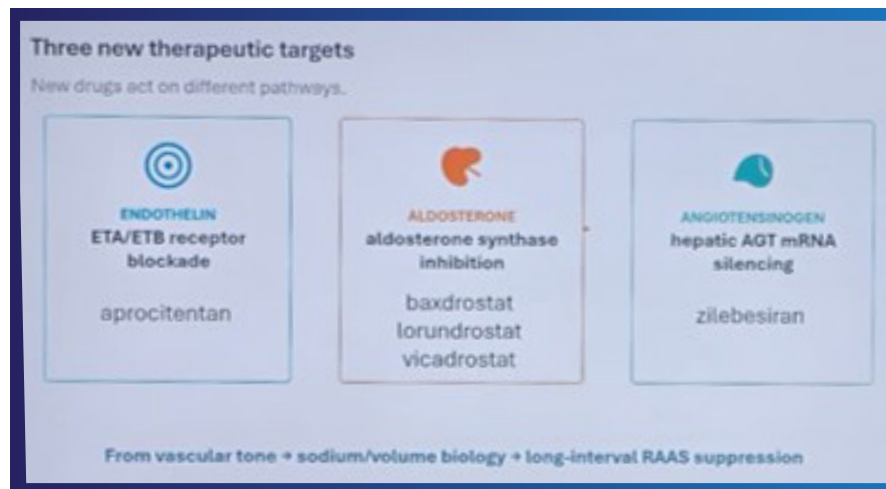
Secondary MI results from an acute condition causing myocardial oxygen supply–demand imbalance. Diagnosis requires acute myocardial injury with symptoms or ECG evidence of ischaemia. Confirmation requires obstructive CAD without acute coronary pathology and/or a new regional wall-motion abnormality or loss of myocardial viability.

Procedure-related MI occurs as a complication of a cardiac procedure within 30 days. Diagnosis is based on evidence of a coronary complication together with a new regional wall-motion abnormality or loss of viable myocardium on imaging; cardiac troponin thresholds are not required. The Fifth UDMI also introduces sex-specific troponin criteria, proposed ICD-11 codes, and updated guidance for myocardial injury and MINOCA.

Conclusion

The Fifth UDMI 2026 provides a simplified, pathophysiology-based classification of MI into primary, secondary, and procedure-related types, with sex-specific troponin criteria improving diagnostic accuracy and reducing potential underdiagnosis in women. These updates aim to standardize diagnosis, guide clinical evaluation, and strengthen research and outcomes assessment.

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Hypertension management is evolving with new therapies targeting endothelin, aldosterone synthesis, and angiotensinogen, alongside the need to confirm true resistant hypertension and individualize treatment according to clinical profile and evidence. The presentation reviewed emerging approaches including Aprocitentan, Zilebesiran, and aldosterone synthase inhibitors (ASIs) such as Baxdrostat, lorundrostat, and Vicadrostat.

Aprocitentan provides dual ETA/ETB receptor antagonism and may be considered in confirmed resistant hypertension, although fluid retention/edema, hemoglobin changes, and teratogenicity require attention. Zilebesiran uses RNA interference to silence hepatic angiotensinogen mRNA, reducing RAAS activity and providing long-lasting blood-pressure lowering with subcutaneous dosing approximately every 6 months. KARDIA-1 and KARDIA-2 demonstrated blood-pressure efficacy and 6-month durability, while KARDIA-3 is evaluating add-on therapy and ZENITH is assessing cardiovascular outcomes in patients with uncontrolled hypertension and cardiovascular disease.

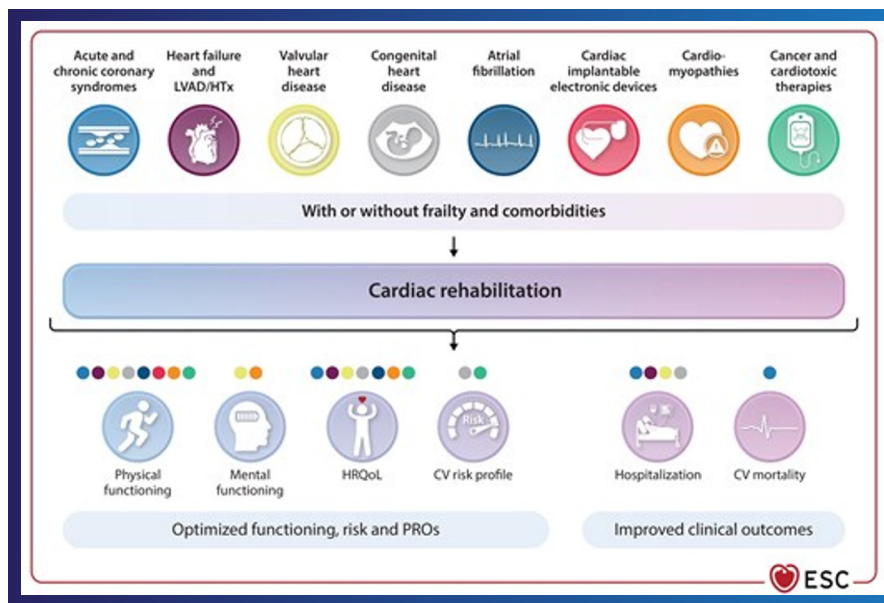
ASIs reduce aldosterone production before receptor activation, with the goal of lowering aldosterone while sparing cortisol synthesis. In ADVANCE-HTN, lorundrostat produced a placebo-adjusted reduction in 24-hour mean ambulatory systolic blood pressure of 7.9 mmHg at week 12 with 50 mg daily; hyperkalemia monitoring was required. Baxdrostat demonstrated a clinically relevant placebo-adjusted seated systolic blood-pressure reduction of approximately 9 mmHg at 12 weeks with 1–2 mg once daily, with potassium and renal function requiring monitoring.

Conclusion

New therapies may provide options for confirmed resistance or uncontrolled hypertension. Before adding treatment, blood-pressure control, treatment optimization, adherence, and secondary causes should be assessed. Drug selection should be guided by patient phenotype, evidence, availability, and monitoring requirements, including potassium, eGFR, edema/volume status, and pregnancy risk.

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Benefits of cardiac rehabilitation for different indications



The 2026 ESC Guidelines emphasize cardiac rehabilitation (CR) as a multidisciplinary, patient-centred intervention across the continuum of cardiovascular care, aiming to improve clinical outcomes, physical and mental functioning, health-related quality of life (HRQoL), and long-term self-management. The guideline promotes early, personalized and accessible CR, with shared decision-making and assessment of patient-reported outcomes (PROs).

Early initiation of CR is recommended for eligible patients to improve enrolment and functional outcomes (Class I, Level C); in hospitalized patients with decompensated HF, early CR should be considered once sufficiently stabilized (Class IIa, Level B2). After ACS, CR should ideally start within 14 days and no later than 30 days; after CABG, within 28 days and no later than 42 days. For ACS/CCS, exercise-based CR reduces cardiovascular mortality, MI and all-cause hospitalization (Class I, Level A) and improves VO₂peak, HRQoL and mental functioning (Class I, Level B1). In chronic HFrEF, CR reduces hospital admissions and improves VO₂peak and HRQoL (Class I, Level B1); similar recommendations apply to HFpEF for improving functional capacity and HRQoL (Class I, Level B1). CR is also recommended for selected patients with congenital heart disease, ICD/CRT recipients, and cancer/cardiotoxic therapy populations, while pulmonary arterial hypertension may be considered (Class IIa, Level B1).

Physician endorsement and automatic referral with individual professional discussion are recommended to improve enrolment (Class I, Level B2). Centre-based, home-based, telerehabilitation and hybrid models can facilitate access and adherence, with programmes tailored to patient needs and preferences. CR should not be withheld because of frailty or comorbidities.

Conclusion

The 2026 ESC Guidelines position CR as an integral, personalized component of cardiovascular care, with greater emphasis on early initiation, comprehensive risk-factor management, patient-centred care, and improving participation through flexible delivery models.

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Effect of dapagliflozin on left ventricular mass in patients with kidney failure receiving maintenance hemodialysis: primary results from the DAPA-HD trial

TA Zelniker

Sodium-glucose cotransporter-2 inhibitors (SGLT2i) reduce cardiovascular events across a broad spectrum of kidney function, including advanced chronic kidney disease, but whether these benefits persist in patients receiving dialysis remains uncertain. The DAPA-HD trial evaluated the effect of dapagliflozin on cardiac structure and function in patients with kidney failure receiving maintenance haemodialysis, with treatment effects assessed according to residual urine output. This session was presented by TA Zelniker at the European Society of Cardiology (ESC) Congress 2026, held from 28th to 31st August 2026 in Munich, Germany.

This academic, multicenter, randomized, double-blind, placebo-controlled trial enrolled patients with kidney failure receiving maintenance haemodialysis and left ventricular hypertrophy. Participants were randomized 1:1 to dapagliflozin 10 mg once daily or placebo for 6 months, stratified by residual urine volume (>200 mL/24 h vs. ≤ 200 mL/24 h). The primary endpoint was change in left ventricular mass index (LVMI), adjusted for body surface area measured by echocardiography performed immediately after dialysis and analyzed using ANCOVA adjusted for baseline LVMI.

A total of 220 patients were randomized (mean age 65 years; 26% female; 42% with T2DM; LVEF 57%; 28% anuric). Baseline LVMI was similar between groups (dapagliflozin: 136 ± 35 g/m²; placebo: 140 ± 38 g/m²); 142 of 210 patients had evaluable 6-month echocardiographic data. At 6 months, LVMI increased by 4.5 ± 25.0 g/m² with dapagliflozin and 8.0 ± 20.6 g/m² with placebo, with no significant between-group difference (adjusted mean difference -3.7 g/m²; 95% CI -11.2 to 3.8 ; $p=0.33$). However, among patients with urine output >200 mL, dapagliflozin reduced LVMI versus placebo (adjusted mean difference -9.2 g/m²; $p=0.035$), with a significant interaction by residual urine output ($p=0.025$).

Conclusion

In patients receiving maintenance haemodialysis, dapagliflozin did not reduce LVMI overall. However, residual urine output significantly modified treatment response, with reverse cardiac remodeling observed only in patients with preserved urine output, suggesting a potential role of residual kidney function in mediating structural cardiac benefits of SGLT2i in dialysis and provide mechanistic insight into SGLT2i biology.

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