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Geriatric Care

Geriatric Care 2026; 12:11708
doi:10.4081/gc.2026.11708

Heart failure patients: what does it change in the elderly?

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Abstract

Heart failure (HF) represents a growing public health challenge, particularly among older adults. Its prevalence increases with age and is frequently complicated by frailty, multimorbidity, and functional decline, all of which worsen prognosis and complicate management. The relationship between HF and frailty is bidirectional: HF promotes sarcopenia, cachexia, and inflammation, while frailty reduces resilience and tolerance to therapy.

The objective of the paper is to summarize current evidence on the management of HF in frail older adults, highlighting recent pharmacological advances, geriatric considerations, and emerging multidisciplinary strategies.

A narrative synthesis has been performed, including the most recent European Society of Cardiology and American Heart Association/American College of Cardiology/Heart Failure Society of America guidelines, position papers, and randomized controlled trials focusing on drug therapy, non-pharmacological interventions, and comprehensive geriatric care in older adults with HF. Guideline-directed medical therapy for HF with reduced ejection fraction—comprising sodium-glucose co-transporter 2 (SGLT2) inhibitors, angiotensin receptor-neprilysin inhibitors, β-blockers, and mineralocorticoid receptor antagonists—remains the cornerstone of treatment. Evidence for HF with mildly reduced ejection fraction and HF with preserved ejection fraction supports SGLT2 inhibitors and individualized management of comorbidities. Multidimensional interventions, including nutritional support, tailored physical rehabilitation, cognitive and psychological care, and telemonitoring, significantly improve outcomes. Structured transitional care and early palliative integration reduce readmissions and enhance quality of life.

Effective management of HF in frail older adults requires combining evidence-based pharmacological therapy with a holistic, geriatric, and multidisciplinary approach spanning hospital, transitional, and community settings. Strengthening randomized evidence and integrating comprehensive care models are essential to improving prognosis and functional independence in this vulnerable population.

Key words: heart failure, frailty, left ventricular ejection fraction.

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Epidemiology

Heart failure (HF) affects over 64 million people worldwide and is primarily a disease of older adults in developed countries. Although HF incidence has declined, its prevalence has increased due to population aging and therapeutic advances.[1] In older adults, the mean prevalence is 8.3%, reaching ~20% among individuals >75 years.[2] The very old (>85 years) show rising incidence despite global declines.[1] HF remains the leading cause of hospitalization in patients >65 years in Western countries, with a prevalence of 11%.[2]

Definition, classification, and diagnosis

HF definitions vary across the literature and practice. The most accepted definition of HF is a clinical syndrome of cardinal symptoms and signs, often with elevated natriuretic peptides, resulting from structural or functional cardiac abnormalities that cause elevated intracardiac pressures and/or inadequate output at rest or during exertion.[3] HF is classified as acute de novo or chronic.[3] International staging describes a continuum from "at risk" to "pre-HF", symptomatic HF, and "advanced HF".[4] Diagnostic scores, however, are seldom validated in the elderly. Optimal

thresholds for natriuretic peptides in those >75 years remain uncertain.[4] According to the European Society of Cardiology (ESC), advanced HF refers to persistent symptoms despite optimal therapy(optimal medical, surgical, and device therapy) (Table 1).[5]

Heart failure in the elderly

HF in older adults reflects cumulative risk factors, such as hypertension, obesity, type 2 diabetes (T2DM), coronary artery disease (CAD), and valvular disease, accelerating cardiovascular (CV) aging and left ventricular (LV) remodeling.[4] Current classifications define HF with reduced ejection fraction (HfrEF) (≤40%), HF with mildly reduced ejection fraction (HfmrEF) (41-49%), and HF with preserved ejection fraction (HfpEF) (≥50%).[4] Elderly HF presentation is often atypical (confusion, cachexia, anorexia, fatigue).[5] Natriuretic peptides are essential for diagnosis and prognosis, though age-adjusted thresholds are needed.[4] Echocardiography remains central; the HfpEF (HFA-PEFF) score integrates imaging and natriuretic peptides.[6] LV filling pressure measurement *via* catheterization is rarely used. Cardiac magnetic resonance imaging adds structural detail, including fibrosis, iron overload, and infiltration, and is a good alternative in patients with poor acoustic windows (such as for obesity or pulmonary disease).[7]

Common acute triggers in HFrEF include coronary syndromes, valvular dysfunction, myocarditis, toxin-induced cardiomyopathy, and anemia.[8] HFmrEF shares atrial fibrillation prevalence and high blood pressure levels with HFpEF, but other features (age and history of chronic kidney disease, diabetes mellitus, and ischemic heart disease) with HFrEF. Transthyretin amyloidosis (ATTR) is now recognized in up to 15% of elderly patients with aortic stenosis or HFpEF.[9] Early recognition *via* serum and urine testing in elderly patients with red flags (symmetric polyneuropathy, HFpEF, aortic stenosis, lumbar spinal stenosis) is essential for targeted therapy.[10] Artificial intelligence (AI) predictive tools, based on routine clinical characteristics, have demonstrated the ability to identify individuals at higher risk of developing HF.[11]

Treatment: new evidence

Older adults were underrepresented in major randomized controlled trials (RCTs), and <40% of frail HF patients receive full guideline-directed medical therapy (GDMT), and most on suboptimal doses. Current management relies largely on post hoc analyses and clinical judgment. Nonetheless, elderly patients should not be excluded from GDMT, provided careful frailty assessment, comorbidity evaluation, and slow drug titration are ensured.[12] Frailty is characterized by a decrease in physiological reserve during stress, constituting a state of vulnerability that entails a higher risk of adverse events. It affects >70% of HF patients ≥70 years and is determined by a bidirectional process: HF worsens frailty through sarcopenia and chronic inflammation, while frailty increases hospitalization and 30-day mortality risk in chronic HF.[13] Management must also address malnutrition, cognitive decline, and functional loss. Furthermore, as recently recommended by an American Heart Association (AHA) statement, other outcomes beyond the traditional ones should be prioritized in older patients with cardiac disease (assessment of quality of life and progression of disability or cognitive impairment).

Guideline-directed pharmacological therapy

HF classification by LV ejection fraction (LVEF) is fundamental for prognosis and treatment evaluation: evidence is strongest for HFrEF, where GDMT includes four core drug classes (Table 2):

i) renin-angiotensin system inhibition: angiotensin-receptor-neprilysin inhibitors, angiotensin-converting enzyme inhibitors (ACEi), or angiotensin (II) receptor blockers (ARB) alone. The PARADIGM-HF trial confirmed sacubitril/valsartan efficacy even in elderly patients;[14]

ii) β-blockers (bisoprolol, carvedilol, nebivolol) reduce mortality and hospitalization. In the SENIORS trial, nebivolol improved outcomes in patients ≥70 years regardless of EF;[15]

iii) mineralocorticoid receptor antagonists (MRAs): in patients with HFrEF, MRAs, such as spironolactone and eplerenone, significantly reduce morbidity and mortality and are recommended as core components of GDMT;[5]

iv) sodium-glucose cotransporter-2 inhibitors (SGLT2i): in patients with HFrEF, regardless of the presence or absence of diabetes, dapagliflozin reduced the risk of worsening HF or death from CV causes.[16]

Additional treatments

Additional agents include diuretics for congestion (not mortality), with diuretic resistance common in the frail elderly,[17] and intravenous iron, which improves symptoms and functional

Table 1. Criteria of advanced heart failure reported by the European Society of Cardiology and American College of Cardiology Foundation/American Heart Association.

Criteria to assess advanced heart failure, ESC criteria	ACCF/AHA criteria
Severe and persistent symptoms of HF (NYHA class III or IV)	Repeated (≥2) hospitalizations or emergency department visits for HF in the past year
Severe cardiac dysfunction defined by a reduced LVEF (≤30%), isolated RV failure or non-operable severe valve abnormalities, or congenital abnormalities, or persistently high (or increasing) BNP or NT-proBNP values and data of severe diastolic dysfunction, or LV structural abnormalities according to the ESC definition of HFpEF and HFmrEF	Progressive deterioration in renal function (*e.g.*, rise in BUN and creatinine). Intolerance to ACEi due to hypotension and/or worsening renal function
Episodes of pulmonary or systemic congestion requiring high-dose intravenous diuretics (or diuretic combinations) or episodes of low output requiring inotropes or vasoactive drugs, or malignant arrhythmias causing >1 unplanned visit or hospitalization in the last 12 months	Weight loss without other causes (*e.g.*, cardiac cachexia)

ESC, European Society of Cardiology; ACCF, American College of Cardiology Foundation; AHA, American Heart Association; HF, heart failure; NYHA, New York Heart Association; LVEF, left ventricular ejection fraction; RV, right ventricular; BNP, brain natriuretic peptide; NT-proBNP, N-terminal pro–B-type natriuretic peptide; LV, left ventricle; BUN, blood urea nitrogen; HfpEF, heart failure with preserved ejection fraction; HfmrEF, heart failure with mildly reduced ejection fraction; HF, heart failure; ACEi, angiotensin-converting enzyme inhibitors.

status.[18] Finerenone, a novel non-steroidal MRA, benefits patients with T2DM and chronic kidney disease and may suit frail individuals intolerant to traditional MRAs.[19] Vericiguat can be added in worsening HF, though caution is warranted in hypotensive or renally impaired patients.[20]

Heart failure with mildly reduced ejection fraction (left ventricular ejection fraction 41-49%)

Evidence is more limited than in HFrEF. ACEi, ARB, β-blockers, MRAs, and sacubitril-valsartan may be considered.[5] The DELIVER trial demonstrated SGLT2i benefit across this spectrum.[21] Guidelines thus mirror HFrEF recommendations, though with lower (class II) evidence levels.

Heart failure with preserved ejection fraction (left ventricular ejection fraction ≥50%)

HFpEF predominates in older adults (>70 years).[22] In this group, data on outcome improvement remain inconsistent. The ESC and AHA/American College of Cardiology/Heart Failure Society of America (ACC/HFSA) guidelines now give SGLT2i class I-IIa recommendations.[10] In EMPEROR-preserved, empagliflozin reduced CV death and HF hospitalization with consistent benefit across age groups, including ≥80 years.[23] Other drug classes may treat comorbidities but show limited prognostic benefit.[10] Frailty is highly prevalent in patients with worsening HF, affecting >50% of patients hospitalized for HF.[8] Such patients with multiple readmissions for decompensated HF have markedly poor outcomes. Given high frailty and multimorbidity, management should emphasize symptom control, comorbidity optimization, and functional maintenance through individualized multidisciplinary care.

Metabolic modulation is emerging: semaglutide aids obesity control but may affect muscle mass; gastrointestinal side effects must be monitored in frail or malnourished elders.[24]

Disease-specific and interventional management

Recent advances have transformed HF care in older adults. Amyloidosis is no longer untreatable—tafamidis reduced mortality and hospitalizations in transthyretin amyloid cardiomyopathy (ATTR-ACT trial).[10,12] Inflammatory myopericardial syndromes may benefit from β-blockers and colchicine.[25]

Non-pharmacological interventions remain critical. Complete revascularization is not universally beneficial in older patients with CAD; individualized decisions are essential. Implantable devices,

Table 2. Pharmacological therapies recommended for heart failure with reduced ejection fraction, heart failure with mildly reduced ejection fraction, and heart failure with preserved ejection fraction (European Society of Cardiology 2021 primary grading).

Drug class/interventions	HFrEF (LVEF≤40%)	HfmrEF (LVEF40-49%)	HfpEF (LVEF≥50%)
ARNi (sacubitril/valsartan)	Class I, level B (preferred to ACEi (if tolerated)	Class IIb, level C (may be considered)	Class IIb, level B (possible reduction in HF hospitalization)
ACEi	Class I, level A (if ARNi not feasible)	Class IIb, level C	(No proven mortality benefit; for comorbidity management)
ARB	Class I, level B (if ACEi not tolerated)	Class IIb, level C	Class IIb, level B (modest effect on HF hospitalization)
β-blockers-BB (bisoprolol, carvedilol, metoprolol succinate)	Class I, level A	Class IIb, level C	Recommended for comorbidities (AF, IHD), not for HF outcomes
MRA (*e.g.*, pironolactone)	Class I, level A	Class IIb, level C	Class IIb, level C (possible reduction in HF hospitalization)
SGLT2 inhibitors (dapaglifozin, empaglifozin)	Class I, level A (irrespective of diabetes)	(evidence emerging)	(ESC 2021 not yet graded)
Loop±thiazide diuretics	Class I, level C (or congestion relief)	Class I, level C	Class I, level C
Ivabradine (sinus rhythm, HR≥70 bpm despite BB)	Class IIa, level B (reduces HF hospitalization)	/	/
Vericiguat (worsening HF despite GDMT)	Class IIb, level B	/	/
Hydralazine/isosorbide dinitrate	Class IIb, level B (for ACEi/ARB/ARNi intolerance)	/	/
Digoxin	Class IIb, level B (lowers hospitalization and no mortality effect)	/	/
Intravenous iron (ferric carboxymaltose, ferric derisomaltose)	Class IIa, level A (improves QoL, functional capacity)	Class IIa, level A (by extrapolation)	Class IIa, level A (by extrapolation)

HfrEF, heart failure with reduced ejection fraction; HfmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HF, heart failure; ARNi, angiotensin receptor-neprilysin inhibitor; ACEi, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; AF, atrial fibrillation; IHD, ischemic heart disease; MRA, mineralcorticoid receptor antagonists; SGLT2i, sodium-glucose cotransporter-2 inhibitors; GDMT, guideline-directed medical therapy; QoL, quality of life.

such as an implantable cardioverter-defibrillator, and cardiac resynchronization therapy remain indicated in EF<35%, left bundle branch block >150 ms, or refractory New York Heart Association III-IV HF, though the benefit varies.[26,27] Transcatheter aortic valve implantation is preferred in ≥70-year-old patients with aortic stenosis, while mitral transcatheter edge-to-edge repair offers an option for high-risk mitral regurgitation. Age alone is not a contraindication for transplantation.[28]

Comprehensive management strategies

Beyond the pharmacological and non-pharmacological approaches already discussed, HF management in frail older adults requires a holistic, multidisciplinary model addressing medical, functional, and psychosocial domains.[5,10]

Key domains of this multidimensional approach include: i) nutrition – correction of malnutrition and sarcopenia through personalized dietary plans; ii) physical rehabilitation – tailored aerobic and resistance programs to improve strength, endurance, and functional capacity, combined with education on disease management; iii) cognitive and psychological support – systematic screening for depression, mild cognitive impairment, and dementia; iv) functional assessment – evaluation of activities of daily living and instrumental activities to maintain autonomy and prevent disability.[13]

To operationalize these principles, a structured geriatric model of care should be integrated across all healthcare settings.

In-hospital phase: all older adults with HF should undergo frailty screening and a comprehensive geriatric assessment to guide GDMT, identify potential deprescribing opportunities, and initiate discharge planning within 72 hours, including rehabilitation, nutrition, physiotherapy, and social services.[29]

Transitional phase: after discharge, structured transitional care, comprising early follow-up within 7 days, HF nurse or telemonitoring contact, medication reconciliation, and education reduce readmissions and emergency visits.[30]

Cardiac rehabilitation: physical rehabilitation, as previously described, improves exercise tolerance, quality of life, and prognosis.[5,13]

Community phase: long-term management should prevent/reduce acute HF exacerbations, monitor therapeutic needs (diuretic needs, iron status, and GDMT adherence), vaccination (influenza and pneumococcal prophylaxis), and check cognition, nutrition, and falls risk, ideally supported by telemedicine and wearable devices, to track vitals to detect early deterioration.[31]

Palliative care should be integrated earlier than at the end of life, whenever there is advanced HF, recurrent worsening episodes, or poor tolerance to GDMT. This optimizes symptom control, caregiver support, and advanced care planning, while hospice referral is appropriate when survival is <6 months.[5,13]

These models reflect ESC (2021) and AHA/ACC/HFSA (2022) recommendations, HFA-ESC frailty position paper, and growing RCT-level evidence supporting transitional, telemedical, and multidisciplinary care models to improve outcomes.[13,16]

Telemedicine in heart failure management

Telemedicine improves continuity and monitoring, particularly in frail patients with limited mobility. The TIM-HF2 trial showed that structured telemedical care reduced CV hospitalization and death.[31] Remote monitoring devices, virtual consultations, and symptom-reporting apps enhance early detection and adherence. Benefits include early decompensation recognition, personalized care, and reduced travel burden. Barriers include limited digital literacy, connectivity, and privacy concerns. Emerging AI and wearable technologies may soon predict exacerbations and optimize therapy in real time.

Conclusions

HF remains a global challenge, particularly in aging populations. Despite advances in pharmacologic and interventional therapy, frailty and multimorbidity continue to complicate management. Integrating evidence-based HF treatment with geriatric expertise in frailty, nutrition, function, and cognition is essential to improve outcomes.

A holistic, patient-centered continuum of care, from acute stabilization to home-based follow-up and palliative support, represents the cornerstone of modern HF management. Strengthening RCT evidence focused on frail elderly patients should be a research priority to develop tailored, multidisciplinary strategies capable of improving survival, function, and quality of life in this complex population.

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Received: 2 September 2023; Accepted: 9 January 2026.

Contributions: all the authors participated in the preparation of the manuscript.

Conflict of interest: the authors declare no potential conflict of interest.

Ethics approval and consent to participate: not applicable.

Availability of data and materials: not applicable.