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Developing a contemporary community clinic for patients with heart failure with preserved ejection fraction within the current National Health Service model

Patrick Tran ,[1,2] Thomas Long,[1] Jessica Smith,[1] Michael Kuehl,[1,3] Tarek Mahdy,[1] Prithwish Banerjee [2,3,4]

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[1]Cardiology Department, University Hospitals Coventry and Warwickshire NHS Trust, Coventry, UK
[2]Centre for Sport, Exercise & Life Sciences, Faculty of Health and Life Sciences, Coventry University, Coventry, UK
[3]Warwick Medical School, University of Warwick, Coventry, UK
[4]Department of Cardiology, University Hospitals Coventry and Warwickshire NHS Trust, Coventry, UK

Correspondence to
Professor Prithwish Banerjee; Prithwish.Banerjee@uhcw.nhs.uk

ABSTRACT

Introduction The diagnostic and therapeutic arsenal for heart failure with preserved ejection (HFpEF) has expanded. With novel therapies (eg, sodium-glucose co-transporter 2 inhibitors) and firmer recommendations to optimise non-cardiac comorbidities, it is unclear if outpatient HFpEF models can adequately deliver this. We; therefore, evaluated the efficacy of an existing dedicated HFpEF clinic to find innovative ways to design a more comprehensive model tailored to the modern era of HFpEF.

Methods A single-centre retrospective analysis of 202 HFpEF outpatients was performed over 12 months before the COVID-19 pandemic. Baseline characteristics, clinic activities (eg, medication changes, lifestyle modifications, management of comorbidities) and follow-up arrangements were compared between a HFpEF and general cardiology clinic to assess their impact on mortality and morbidity at 6 and 12 months.

Results Between the two clinic groups, the sample population was evenly matched with a typical HFpEF profile (mean age 79±9.6 years, 55% female and a high prevalence of cardiometabolic comorbidities). While follow-up practices were similar, the HFpEF clinic delivered significantly more interventions on lifestyle changes, blood pressure and heart rate control ($p<0.0001$) compared with the general clinic. Despite this, no significant differences in all-cause hospitalisation and mortality were observed. This may be attributed to the fact that clinic activities were primarily cardiology-focused. Importantly, non-cardiovascular admissions accounted for >60% of hospitalisation, including causes of recurrent admissions.

Conclusion This study suggests that existing general and emerging dedicated HFpEF clinics may not be adequate in addressing the multifaceted aspects of HFpEF as clinic activities concentrated primarily on cardiological measures. Although the small cohort and short follow-up period are important limitations, this study reminds clinicians that HFpEF patients are more at risk of non-cardiac than HF-related events. We have therefore proposed a pragmatic framework that can comprehensively deliver the modern guideline-directed recommendations and management of non-cardiac comorbidities through a multidisciplinary approach.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ To improve the prognosis of patients with heart failure with preserved ejection (HFpEF), a holistic and patient-centred approach is needed, with a focus on cardiac and non-cardiac comorbidities.
⇒ There is a lack of data on whether existing clinic models can adequately deliver this care.

WHAT THIS STUDY ADDS

⇒ No significant differences in all-cause hospitalisation and death were found between the dedicated HFpEF and general cardiology clinic cohorts.
⇒ An evaluation of the existing clinic models revealed that clinic interventions were primarily cardiology focused with a lack of measures on non-cardiac comorbidities, even though non-cardiac hospitalisation accounted for >60% of admissions, emphasising that HFpEF patients are more at risk of non-cardiac than HF-related events.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Consistent with several national initiatives, we propose a practical and personalised outpatient model, tailored towards HFpEF, which can comprehensively deliver the modern guideline-directed recommendations that include the prescription of novel sodium-glucose co-transporter 2 inhibitors and management of non-cardiac comorbidities through a multidisciplinary approach.

INTRODUCTION

The therapeutic landscape of heart failure with preserved ejection fraction (HFpEF) has recently shifted. Its clinical trajectory can now be ameliorated by the expanding role of sodium-glucose co-transporter 2 inhibitors (SGLT2i).[1] From its pleomorphic effects on arterial stiffness, cardiometabolism and renal function, physicians are reminded that HFpEF is a heterogeneous syndrome

driven by an interplay of cardiovascular and non-cardiac comorbidities.[2–4] SGLT2i is not the panacea for HFpEF. The exclusion of patients with significant comorbidities, cardiac amyloidosis, absence of overall mortality benefit coupled with significantly high non-cardiovascular deaths and hospitalisation in the EMPEROR-PRESERVED and DELIVER trials,[5] remind us that HFpEF care must still be systematic, individualised and holistic.[1 6]

One individualised approach is to target specific therapies to specific phenotypes, but this also has its limitations. In principle, the ageing phenotype may guide strategies for reducing vascular stiffness[7]; an obesity phenotype may permit the use of glucagon-like peptide-1 receptor agonists (GLP1-RA) by weight management specialists,[8] while a renal dysfunction phenotype may prompt ultrafiltration or SGLT2i for renal protection regardless of HF.[9–11] In practice, the marked overlap between groups makes this approach difficult.[12] With the array of diagnostic intricacies, novel therapies and firmer recommendations to optimise non-cardiac comorbidities,[12 13] it is uncertain if our current clinic model can adequately deliver this to the HFpEF population.

The evidence that multidisciplinary clinics reduce HF hospitalisations have so far been limited to patients with HF with reduced ejection fraction (HFrEF).[12–15] Hence, there have been growing calls by professional bodies to develop an outpatient model tailored towards HFpEF.[12 13] We have previously suggested that a collaborative cardiogeriatric clinic may provide better care for patients with HFpEF through an all-round approach to comorbidity and frailty.[16] While the concept of joint clinics seems appealing, it is unlikely to serve as a practical solution within the finite resources of the National Health Service (NHS). Based on our experiences in running a multispecialty cardiorenal clinic,[10] which serves a smaller patient group, the financial costs and shortage of specialists make it difficult to translate this model to the sheer population size of HFpEF.[2 3] Instead, outpatient HF pathways should be redesigned into a more efficient system. In line with the NHS long-term plan and Getting it Right First Time (GIRFT) report for cardiology,[17 18] the standard HF clinic model needs innovative transformation. Before this can be accomplished, we must first ascertain the efficacy of the current HFpEF clinic model, for which data on this is lacking.

University Hospitals Coventry and Warwickshire (UHCW) NHS Trust was one of the first UK centres to establish a dedicated HFpEF community clinic, which was set up before the advent of novel cardiometabolic therapies.[1 19] Instead, the main pillars of HFpEF management consisted of education, exercise, volume control, management of blood pressure and atrial fibrillation (AF).[20 21] It is important to appreciate that when this clinic was conceived, there was neither a structured, funded provision of care, nor HF specialist nurse (HFSN) support, unlike the HFrEF service. Since no studies have appraised the efficacy of this HFpEF clinic model, we performed a retrospective evaluation to ascertain whether such interventions had any impact on patient outcomes, compared with those treated in a general cardiology clinic. It was anticipated that if efficacy was not shown in this study, a contemporary clinic pathway would be proposed in the discussion of this article.

METHODS

Study setting

UHCW is a tertiary cardiac centre that runs daily consultant-led general cardiology clinics, where a mixture of patients with HFrEF and HFpEF are seen by both HF and non-HF specialists including trainee registrars. Prior to the global viral pandemic when all consultations were held face to face, a specific HFpEF community clinic was set up, which was solely led by a consultant cardiologist specialising in HF in the City of Coventry Health Centre, UK. The dedicated HFpEF clinic was a smaller clinic which saw approximately up to 6 suspected HFpEF patients every week as opposed to the general clinic which was a larger clinic of around 24 patients seeing a mixture of HF patients of all types including HFpEF. As the latter clinic was busier, not all patients seen by cardiology registrars in that clinic were necessarily reviewed or discussed with the consultant cardiologist. Furthermore, the dedicated HFpEF clinic was also involved in recruiting patients into research trials, for example, the earlier PARAGON study.

Study design

In this single-centre retrospective study, clinic activities from the dedicated HFpEF clinic were recorded and examined whether such interventions had any prognostic effects on cardiovascular and non-cardiovascular mortality and unplanned admissions, when compared with patients managed in a general cardiology clinic. The diagnosis of HFpEF was defined by the presence of HF symptoms±signs, left ventricular ejection fraction (LVEF) ≥50% with morphological and/or functional cardiac abnormalities associated with correlates of raised left ventricular filling pressures, for example, elevated E/e' or BNP.[12 22] E/e' was not available in patients with significant mitral regurgitation, mitral annular calcification or unreliable measurements of E velocity. Given the nascent stages of the HFpEF clinic, 101 patients were consecutively analysed and compared with a random selection of 101 patients with HFpEF from the general clinic. Data were collected over a 12-month period when the HFpEF clinic was set up in 2017 and follow-up was evaluated until 2019 before the COVID-19 pandemic. No criteria were used to match the two populations. In terms of the duration of HFpEF, based on symptom duration, this was an average 7 months±0.3 (SEM). Since National Institute for Health and Care Excellence (NICE) guidance (NG106) for HF referral was followed, we can assume that the interval between presentation to general practitioners and review at the outpatient clinic was either 2 weeks if NT-pro B-type natriuretic peptide (BNP) was above 236

pmol/L (>2000 ng/L) or 6 weeks if between 47 and 236 pmol/L (400–2000 ng/L).

Sources of referral

All HFpEF patients from both clinics were based on the same referral criteria set out by the NICE guidance (NG106) described above. Sources of referral were from general practice, secondary care and the local echocardiography service whenever HFpEF was suspected on an echocardiogram. Referrals by secondary care were also made via electronic discharges where the instruction to refer for HF follow-up was actioned by the non-clinical bookings team without necessarily differentiating between the specialised HFpEF and general clinic. Furthermore, due to the lower availability of the HFpEF clinic than the general clinic, not every HFpEF patient would be seen in the specialised clinic in an acceptable timeframe and would therefore be reviewed in the latter clinic. In other words, patients who were seen in the HFpEF clinic occurred at random and depended on clinic appointment availability. This is one important reason why the British Society of HF has recently called for greater resource and workforce expansion to address this shortcoming.[23]

Data collection

Electronic and paper records were used to extract patient demographics, comorbidities, medications, New York Heart Association (NYHA) functional class at the time of the index consultation, followed by the number of follow-ups over the study period. Data collection occurred before SGLT2i were approved for use in HF and cardiovascular outcome improvement, and thus were not collected at baseline. Clinic activities (eg, medication changes, management of volume status/hypertension/heart rate) were recorded to determine the scope of interventions that were cardiac and non-cardiac orientated. The number and causes of unplanned HF-related and non-cardiovascular admissions (first and recurrent), all-cause and cardiovascular-related deaths at 6–12 months from the time of the first clinic visit were collected using electronic discharge summaries. Causes of rehospitalisation (>1 admission since the index hospitalisation over the 12-month study period) were also recorded.

Statistical analysis

Continuous data, reported as mean (SD) or median (IQR), were compared using Student's t-test or Mann-Whitney U test, depending on normality distribution. Categorical data, summarised by count (percentages), were compared using χ^2 test or Fisher's exact test. A $p<0.05$ was considered statistically significant. SPSS V.28.0 (IBM) was used for analysis.

RESULTS

Baseline characteristics

The clinical profile of patients with HFpEF is summarised in table 1. Expectedly, they were generally older,

Table 1 Baseline characteristics of patients

	HFpEF clinic (n=101)	General clinic (n=101)	P value
Age—year (mean±SD)	78±9.8	79±9.4	0.38
Female sex—no (%)	57 (56.4)	55 (54.5)	0.89
Comorbidities—no (%)			
Hypertension	78 (77.2)	79 (78.2)	0.87
Coronary artery disease	28 (27.7)	33 (32.7)	0.44
Atrial fibrillation	51 (50.5)	56 (55.4)	0.48
Type 2 diabetes	31 (30.7)	36 (35.6)	0.46
Valvular heart disease	25 (24.8)	20 (19.8)	0.40
Chronic kidney disease‡	38 (37.6)	44 (43.6)	0.39
Chronic obstructive pulmonary disease	11 (10.9)	11 (10.9)	1.00
Obesity (BMI≥30)	17 (16.8)	10 (9.9)	0.15
NYHA functional class—no (%)			
I	5 (5.0)	6 (5.9)	1.00
II	53 (52.5)	42 (41.6)	0.17
III	39 (38.6)	53 (52.5)	0.07
IV	4 (4.0)	0	0.12
NT-pro B-type natriuretic peptide (mean±SEM)	367±56	431±65	0.46
Echocardiographic data			
Left ventricular hypertrophy—no (%)	28 (27.7)	35 (34.7)	0.29
LVEF (mean±SD)	52.5±7.0	53.7±6.6	0.22
E/e' (mean±SD)*	17.1±5.4	17.2±5.6	0.88
PASP (mean±SD)†	47.4±16.9	42.7±15.8	0.17
Indexed left atrial volume—mL (mean±SD)	56.8±17.0	53.9±12.5	0.49
Type of medications—no (%)			
Beta-blockers	49 (48.5)	63 (62.3)	0.05
RAAS inhibitors	46 (45.5)	47 (46.5)	0.89
MRA	12 (11.9)	7 (6.9)	0.23
Loop diuretics	61 (60.4)	63 (62.4)	0.77
Digoxin	7 (6.9)	13 (12.9)	0.16
Average no of clinic visits, median (IQR)	1 (1–2)	1 (1–2)	0.07
No of clinic visits			
1	68	56	0.12
2	24	29	
3	6	14	
4	3	1	
5	0	1	

*Data based on 78 patients in HFpEF and 63 patients in general clinic.
†Data based on 52 patients in HFpEF and 45 patients in general clinics with measurable tricuspid regurgitation Doppler traces.
‡Based on estimated glomerular filtration rate <60 mL/min/1.73 m^2.
BMI, body mass index; HFpEF, heart failure with preserved ejection; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid; NYHA, New York Heart Association; RAAS, renin–angiotensin–aldosterone system.

predominantly female with a high prevalence of hypertension, AF, type 2 diabetes mellitus (T2DM), chronic kidney disease (CKD) and obesity. No patients with T2DM were prescribed SGLT2i at baseline evaluation. A large proportion reported substantial limitation of physical activity (NYHA III) during their first consultation; approximately 60% were already on oral diuretics. As shown in table 1, baseline echocardiography demonstrated similar proportions of patients with LV hypertrophy, severe left atrial dilatation, average LVEF between 52 and 54%, average E/e' around 17 and pulmonary artery systolic pressures between 42 and 47 mmHg. Based on these characteristics, both sample populations from the HFpEF and general cardiology clinics were evenly matched (no predefined selection or matching criteria were used) and reflected a typical ageing and multimorbid HFpEF profile[7] (table 1). In terms of anticoagulation for patients with AF, there was a similar prescription of direct oral anticoagulants (35.3% vs 25.0%, p=0.245) and vitamin-K antagonists (58.9% vs 46.4%, p=0.200) between the HFpEF and general cardiology clinics. However, significantly more patients from the general clinic were not on oral anticoagulation at baseline (28.6% vs 5.9%, p=0.002) despite being considered appropriate, as we found that they were still awaiting a referral to the outpatient haematology nurse-led clinic for initiation of anticoagulation at clinic evaluation.

Frequency of clinic visits

Since follow-up plays a critical role in achieving better HF outcomes,[24] a comparison of clinic attendances provides insight into the follow-up capacity between the two clinic models. The frequency of consultations per patient was similar, with the majority attending the respective clinics only once over the study period (table 1). Given the weekly availability of HFpEF clinics, it was anticipated that fewer patients would have>1 clinic attendance over 12 months, compared with the general clinic cohort. However, this was not statistically significant (32.7% vs 44.6%, p=0.08), and the distribution of the number of follow-ups was also similar (p=0.12) (table 1). Despite limited appointment availability, patients attending the HFpEF service were still able to be reviewed frequently if clinically indicated, as reflected by three patients who attended four times in 12 months. Notably, the initial consultation time in the HFpEF clinic was at least 30 min long, which is almost twice the time spent in general clinics. Hence, one HFpEF clinic may equate to around two clinic visits in the latter group.

Clinic activities

The service evaluation focused on interventions delivered to patients during their first consultation. In both clinics, it was clear that most interventions were cardiology-focused, namely congestion management (64% patients), review of antihypertensives (86%) and rate or rhythm control of AF with either cardioversion or referral for ablation (48%). Non-pharmacological measures consisted of education on fluid (to ~1.5 L/day)

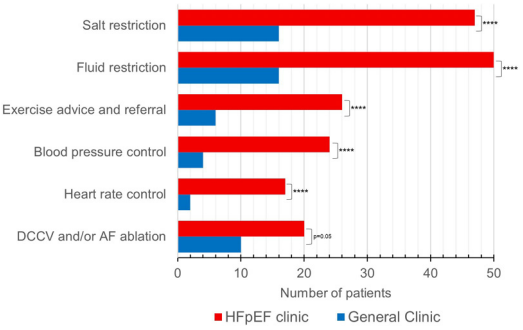

Figure 1 Comparison of non-pharmacological interventions between the two clinics. ****p<0.0001; AF, atrial fibrillation; DCCV, DC cardioversion; HFpEF, heart failure with preserved ejection fraction.

and salt restriction (<2400 mg/day), advice on moderate-intensity exercise according to national guidelines,[25] including referrals to cardiac rehabilitation for personalised exercise programmes. Patients seen in the dedicated HFpEF service were significantly more likely to receive these interventions than those attending the general clinic (p<0.0001) (figure 1).

In terms of pharmacological measures, medication changes were primarily aimed at volume control with oral diuretics, optimisation of BP ≤130/80, and controlling heart rate <70 bpm (in sinus rhythm) or <90 bpm (in AF). The main drugs prescribed were diuretics, beta-blockers, renin–angiotensin–aldosterone system inhibitors (RAASi) and MRA. According to figure 2, both groups displayed similar patterns of medication changes across all drug classes, with a greater tendency to continue with medications unchanged. In both clinics, beta-blockers and diuretics were most frequently initiated, whereas the prescription of RAASi was substantially lower. The indication of RAASi was limited to hypertension, despite the well-established renoprotective effects of angiotensin-converting enzyme inhibitors in CKD[26]: a comorbidity that was present in over one-fifth of both groups. Worsening renal function (estimated glomerular filtration rate <30mL/min/1.73 m^2) would have limited its use.

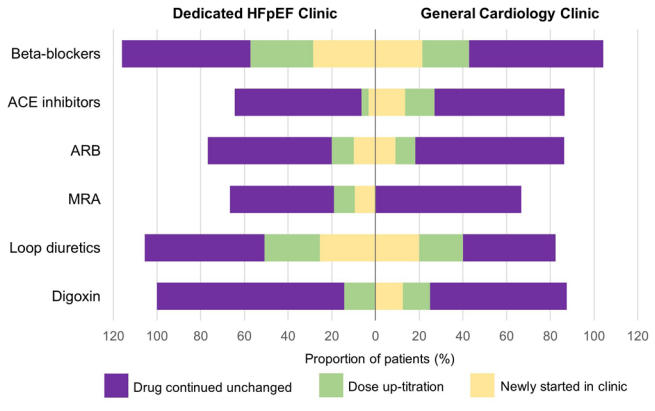

Figure 2 Comparison of medication changes at first clinic consultation. ARB, angiotensin receptor blocker; HFpEF, heart failure with preserved ejection; MRA, mineralocorticoid receptor antagonist.

Table 2 Hospitalisation and mortality at 6 and 12 months

	HFpEF clinic (N=101)	General clinic (N=101)	P value
Primary outcome at 6 months			
Total no of hospital admissions at 6 months	65	53	0.36
Unplanned heart failure admissions—no (%)	21 (32.3)	21 (39.6)	1.00
Non-cardiovascular admissions—no (%)	44 (67.7)	32 (60.4)	0.22
All-cause mortality at 6 months—no (%)	7 (6.9)	3[3]	0.19
Cardiovascular deaths	2	2	1.00
Non-cardiovascular deaths	2	0	0.16
Unknown cause of death	3	1	0.31
Primary outcome at 12 months			
Total number of hospital admissions at 12 months	103	112	0.15
Unplanned heart failure admissions—no (%)	34 (33.7)	45 (40.2)	0.74
Non-cardiovascular admissions—no (%)	69 (68.3)	67 (59.8)	0.84
All-cause mortality at 12 months—no (%)	15 (14.9)	7 (6.9)	0.07
Cardiovascular deaths	3	3	1.00
Non-cardiovascular deaths	8	2	0.03
Unknown cause of death	4	2	0.32
Causes of rehospitalisation (> 1 unplanned admission over 12 months)			
Decompensated heart failure	11	15	0.37
Community-acquired pneumonia	5	4	
Postural hypotension-related falls	2	4	
Symptomatic anaemia	3	2	
Acute kidney injury	5	1	

HFpEF, heart failure with preserved ejection.

Clinical outcomes

Hospitalisation and mortality rates were compared at 6 and 12 months to assess the efficacy of the two service models based on their clinic-based activities and follow-up arrangements. The first important observation was that in both groups, non-cardiovascular hospitalisation accounted for at least 60% of all hospitalisations at 6 and 12 months. Second, no significant differences were found in cardiovascular mortality, HF-related and non-cardiovascular causes of hospitalisation (table 2). There is some suggestion that non-cardiovascular deaths were higher in the HFpEF clinic group (8 vs 2 deaths, p=0.03), of which 50% were due to community-acquired pneumonia. Cause of death was unclear when it was recorded in the community or at another hospital. After adjusting for age, baseline NT-pro-BNP, LVEF, E/e' and major comorbidities (CKD, AF, chronic obstructive pulmonary disease (COPD), obesity, diabetes, hypertension, coronary artery disease, valvular heart disease) in a multivariate logistic regression, no significant differences were observed between the HFpEF and general cardiology clinic groups in all-cause mortality (p=0.90), cardiovascular (p=0.99) and non-cardiovascular deaths (p=0.95).

Of the total hospital admissions, 26 recurrent hospitalisations occurred in both HFpEF (n=26/103, 25.2%) and general clinic groups (n=26/112, 23.2%). The most common reason for rehospitalisation was decompensated HF. Compared with the general clinic cohort, non-cardiovascular presentations contributed to a slightly higher proportion of recurrent admissions in the HFpEF clinic group (57.7% vs 42.3%, p=0.67). In order of frequency, common causes were community-acquired pneumonia, falls from postural hypotension, symptomatic anaemia (from iron deficiency anaemia or complications of anticoagulation for AF) and acute kidney injury from nephrotoxicity or overdiuresis in the presence of infection. These were similar between the two groups (p=0.12).

DISCUSSION

This study is among the first to examine whether a dedicated HFpEF clinic can adequately deliver the holistic, individualised care required to improve patient outcomes in a typical HFpEF population. Three notable findings were observed: (1) patients managed in the dedicated HFpEF clinic had a similar rate of cardiovascular and non-cardiovascular death and hospitalisation compared with patients managed in the general clinic, despite greater input from the former clinic on lifestyle

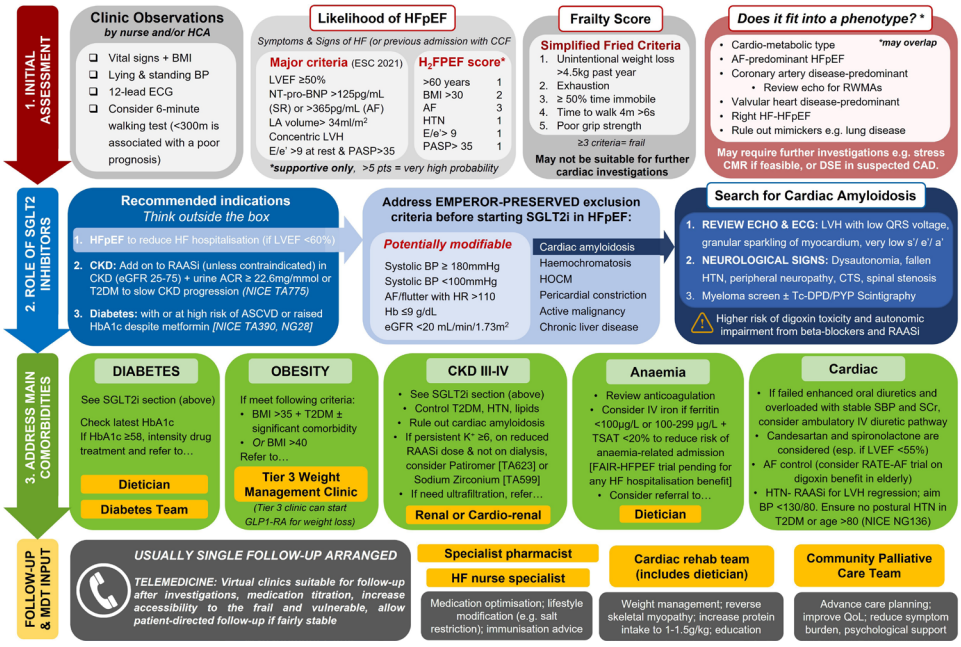

Figure 3 Proposed HFpEF clinic pathway for a comprehensive and holistic approach to HFpEF. AF, atrial fibrillation; BMI, body mass index; CKD, chronic kidney disease; CTS, carpal tunnel syndrome; eGFR, estimated glomerular filtration rate; GLP1-RA, glucagon-like peptide-1 receptor agonist; HF, heart failure; HTN, hypertension; LA, left atrium; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; RAASi, renin–angiotensin–aldosterone system inhibitors; RWMAs, regional wall motion abnormalities; PASP, pulmonary arterial systolic pressure (mmHg); SBP, systolic blood pressure; SCR, serum creatinine; SR, sinus rhythm; T2DM, type 2 diabetes mellitus; TSAT, transferrin saturation.

modification, hypertension and AF control; (2) non-cardiovascular hospitalisation dominated in both clinic groups, similar to that reported in recent HFpEF trials,[27] emphasising that patients with HFpEF are more at risk of non-cardiac than HF-related adverse events and (3) this may be attributed to the third finding that the observed clinic activities, which are likely representative of most HF clinics, were primarily cardiology focused.

Contemporary HFpEF clinic model

Clearly, the current clinic model is not sufficiently structured to deliver the guideline-directed recommendations expected in the modern era of HFpEF. There is thus an imminent need to design a more comprehensive, patient-centred HFpEF clinic that has the capacity to optimise the main cardiac and/or non-cardiac comorbidities.[18] Due to interhospital variations, it would be counterproductive to dogmatically promote a single clinic model, which would not be universally applicable. Instead, we propose a HFpEF clinic pathway, illustrated in figure 3, that incorporates four core themes as general guiding principles: (1) earlier recognition of cardiac amyloidosis (CA) and frailty to guide investigations and treatment, including SGLT2i; (2) greater focus on evidence-based therapies for non-cardiac comorbidities; (3) harnessing the full potential of healthcare assistants, HFSN and clinical pharmacists in pre-clinic assessments, education, medication optimisation and follow-ups and (4) integration with telemedicine and streamlining with ambulatory pathways.

Frailty and HFpEF

Frailty and HFpEF are often intertwined in elderly patients, accompanied by sarcopenia, metabolic dysregulation and deconditioning.[28 29] These factors generally make them unsuitable for further complicated investigations and complex medication regimens (ie, polypharmacy). For these reasons, recognising frailty should form an integral part of initial clinic assessments. As outlined in figure 3, a simplified Fried criterion can be adapted as a practical means of gauging frailty, of which three of the five components (ie, slowness, exhaustion, low physical activity) may be quantified by a 6 min walk test.[30 31] Prognostically, <300 m is associated with increased mortality risk.[31] With a trundle wheel and stopwatch, this can be feasibly assessed by clinic nurses and trained healthcare assistants in a 1–2 m^2 dedicated area. Synchronously, lying and standing or even walking BP measurements can be performed in the elderly or patients with diabetes, as per the NICE hypertension guidelines.[32] Detecting postural hypotension will limit the use of antihypertensives and reduce falls: a common cause of recurrent admissions in our study group.[29]

A multidisciplinary approach can improve or even reverse aspects of frailty, especially if intervened early. Cardiac rehabilitation, which is recommended by NICE (NG106) irrespective of HFrEF or HFpEF, can offer resistant training to counteract skeletal myopathy, peripheral vascular dysfunction and physical deconditioning,[3 33] while dietician-guided nutrition (eg, increasing protein intake to 1–1.5 g/kg/day) may minimise sarcopenic

muscle loss.[12 34] Streamlining funding is required for such an approach in HFpEF patients.[23] For the clinician, deprescribing can reduce adverse drug reactions, which accounts for over two-fifths of hospitalisations in the elderly,[29] and in the severely frail, a discussion on palliation or referral to the palliative care team can direct attention to advance care planning and improving quality of life.[12] A recent observational study on a Spanish HF, which focused on the care of multimorbid elderly HF patients, primarily with HFpEF and led by a general internist, highlighted the importance of early recognition of palliation based on the Charlson Comorbidity Index and the use of the 'surprise question' on the likelihood of death in the next 12 months.[35] This can be prioritised at the outset of the clinic for patients identified as severely frail to facilitate improving quality of life.[36] In contrast to our HFpEF clinic model, which aimed to reduce the risk of hospitalisation and recurrent admissions, patients referred to the Spanish HF unit had to have at least one episode of decompensated HF prior to referral. The study also highlighted the poor prognosis associated with low eGFR but did not suggest how these patients should be specifically managed in clinic. From our experience, some of these patients may be better served in a joint cardio-renal clinic, which we also run alongside the HFpEF clinic.[10]

Practical considerations of SGLT2i in HFpEF

Frailty is not a contraindication to SGLT2i. It was recently found to be associated with improved cognition and gait speed by improving endothelial function and ultimately arterial stiffness.[4 37] Nonetheless, adverse drug reactions can still occur. Hence, individualised advice, drug adjustments and sick day rules must be provided, perhaps by a HFSN or clinical pharmacist. For example, doses of concurrent sulphonylurea or insulin in patients with T2DM and eGFR >45 mL/min/1.73 m^2 should be reduced by 20% to mitigate hypoglycaemia.[38] Diuretics and antihypertensives may need to be downtitrated if baseline systolic BP is ≤100 mm Hg. Apart from advice on genital hygiene to lower the risk of genitourinary infections, any dehydrating acute illnesses should trigger a temporary suspension of SGLT2i to avoid euglycaemic ketoacidosis.[13 38]

In our current practice, SGLT2i is considered in HFpEF if there is a need for better glycaemic control, concurrent or high-risk of atherosclerotic cardiovascular disease in T2DM (NICE NG28), or as a second line to RAASi in CKD stage III–IV with albuminuria (NICE NG203). For HFpEF alone, it is currently considered if there is a history or high risk of HF hospitalisation, providing LVEF is <60% with NYHA II–IV symptoms.[3 27] More importantly, before prescribing SGLT2i for HFpEF as a standalone indication, the exclusion criteria of the EMPEROR-PRESERVED trial must be addressed (figure 3). Modifiable exclusion variables include systolic BP >180 mm Hg, uncontrolled AF and anaemia. Indeed, significantly more patients in the trial were on RAASi (80%), beta-blockers (87%) and MRAs (37%) compared with our clinic cohort, even after accounting for the new prescriptions.[1] This suggests that the real-world population may require more haemodynamic optimisation before starting SGLT2i, including individualised treatment with MRAs based on the TOPCAT-America subgroup analysis in which prognostic signals were detected with spironolactone.[39] By addressing these modifiable parameters, a systematic and holistic approach will naturally be adopted. It will also prompt clinicians to actively search for excluded patient populations, such as haemochromatosis and infiltrative diseases, particularly CA: an underdiagnosed cause of HFpEF in at least 17% of cases.[40 41]

Cardiac amyloidosis

Although hypertension remains the most likely aetiology of HFpEF,[3 7] it is necessary to adopt a structured approach to investigate the increasing number of patients with restrictive cardiomyopathies, especially CA. One important step for the HF physician is to personally review the echocardiogram for signs of infiltration, for example, apical sparing of longitudinal strain or a speckled hypertrophied ventricular septum, supported by an ECG that shows disproportionately low QRS voltages.[41] Dysautonomia and neurological symptoms are also high-yield characteristics (figure 3). All suspected cases should undergo immunofixation electrophoresis, while cardiac MRI and bone scintigraphy can establish the diagnosis, which directly impacts on medical therapy.[12] Apart from preventing SGLT2i initiation, it urges the cautious use of RAASi and beta-blockers which can compound the frequent complication of autonomic dysfunction, as well as digoxin which avidly binds to amyloid fibrils. As shown in our study, these drugs are commonly prescribed preclinic and continued unchanged. The concern of digoxin toxicity may become more relevant as its use in permanent AF may rise following the RATE-AF trial, which found better NYHA improvement and reduction in NT-pro-BNP with digoxin than beta-blockers in an elderly population comparable to our study group.[42]

Adopting an integrated approach to HFpEF

Since the recent reform of UK cardiology training from single to dual accreditation with general internal medicine, cardiologists will inevitably become more confident in managing medical issues associated with HFpEF. That said, it would be unrealistic for a cardiologist to tackle all comorbidities alone in a siloed HFpEF clinic without multispecialty input. Hence, efficient coordination with other specialties is key, either before (eg, implementing a triage process) or conventionally after clinic. To help alleviate workload, common clinic activities such as medication optimisation, congestion control, education on lifestyle factors, immunisation and exercise (which formed the majority of interventions observed in both clinic groups) can largely be delegated to specialist HF nurses and clinical pharmacists, who can independently prescribe within the scope of their practice.[23] Upskilling

and extending the roles of the HF workforce as per the GIRFT initiative will free up capacity for the cardiologist to focus on more complex diagnostic and therapeutic aspects of HFpEF.[18]

In accordance with the NHS long-term plan, UK cardiologists should not only be cognizant of the latest evidence-based therapies for common medical conditions, but also be confident to initiate first-line therapies for specific comorbidities while the referred patient awaits specialist review.[17] For example, newly diagnosed or poorly controlled T2DM can be optimised with metformin, gliptins, SGLT2i or GLP1-RA before the patient attends diabetic specialist review (NICE NG28). HFpEF mimickers such as COPD, confirmed on spirometry (a test that is sometimes requested in HF clinics), can be tried on inhalers recommended by NICE (NG115), pending COPD specialist assessment. For obesity, an awareness of the referral criteria to tier 3 wt management service (figure 3) will provide patients access to weight loss surgery and NICE-approved GLP1-RA e.g. liraglutide (TA664) or semaglutide (GID-TA10765) for effective weight loss.[8] Finally, anaemia-related admissions, another common cause of recurrent admissions in our study cohort, can be mitigated by reviewing antithrombotic therapies and correcting any iron deficiency with intravenous iron, a therapy that may be given in established ambulatory day units, if available.[12 13] It is crucial for HF specialists to get trained up in the basic management of these common comorbidities to initiate these therapies themselves before seeking more specialised help, if needed. Virtual multidisciplinary team (MDT) clinics could be the most efficient, cost-effective way for advanced comorbidity control and reducing HF hospitalisation.[43]

Ambulatory pathways and digital innovation

Ambulatory settings provide an ideal environment for care optimisation and intravenous therapies, for example, diuretics without resorting to unnecessary hospitalisation. The UHCW HF team has previously reported the efficacy and feasibility of a diuretic day-care service for HF patients with fluid overload who were otherwise stable from a haemodynamic and renal standpoint. Patients quickly improved within a few hours, achieving marked weight loss in >80% and circumventing unplanned admissions in 94% of cases.[44] Not only does this reduce hospitalisation costs, but it also abates the risk of further deconditioning from prolonged hospital stays in the frail population.[28] The development of virtual wards as part of the ICS plan is likely to improve this provision quickly.

As demonstrated in our study, there appears to be large variability in follow-up practice, with some electing to follow up all HFPEF patients at least once after the first diagnosis, while others discharge them back to general practitioners with advice. Clinic availability is a limiting factor to regular follow-up that may be improved with telemedicine. With the recent expansion of telemedicine since the viral pandemic, virtual consultations have provided greater accessibility to the vulnerable and have been associated with lower HF hospitalisation.[45 46] Telemedicine is thus emerging as the new normal means of following up patients after investigations and for medication optimisation; it has been readily adopted by our MDT in the outpatient HF service. Finally, as digital innovation develops, devices that can measure pulmonary artery pressure in vivo (based on the CHAMPION and GUIDE-HF studies) or subcutaneous monitors that integrate established algorithms from cardiac implantable electronic device platforms to detect early signs of HF decompensation may become integral in the future routine care of HFpEF.[47]

Simplified (barebone) approach to HFpEF

Since HFpEF patients are generally very elderly, frail, multimorbid with limited mobility, adopting a simplified approach to investigations and treatment is warranted. While a comprehensive ideal approach to our clinic model as a framework for thinking through every patient is proposed, a straightforward approach is often adopted. As illustrated in figure 4, after review of the clinical features of HF, blood tests (including non-invasive screen for AL amyloidosis), ECG and echocardiogram, a clinical judgement is made to decide if further investigations would be appropriate or tolerated. This is often a clinical judgement based on overall 'eyeball' impression. If further investigations are clinically justified (in our practice, this occurs in <50% cases), the standard recommended investigation is a stress cardiac MRI to mainly exclude transthyretin-CA and myocardial ischaemia, while confirming the diagnosis of HFpEF over pure right HF. In those with associated pulmonary hypertension on echocardiography with suspicion of a pre-capillary component, a right±left heart catheterisation is sometimes undertaken. Treatment follows the principles in figure 3 but in a more simplified format as outlined in figure 4.

Limitations

This study should be interpreted within the context of its limitations. First, the retrospective nature cannot infer cause and effect. Although a notable limitation is the small sample size, it is likely a true reflection of the realistic throughput of HFpEF patients attending these types of community clinic due to time and resource constraints. We acknowledge that the small cohort and short follow-up time will reduce the generalisability and reliability of the study findings. A further retrospective analysis of the study population beyond the 12-month follow-up period found that approximately 50% of patients had passed away primarily from COVID-19, which prevented a longer follow-up time. Important lessons from this study should not be ignored based on this limitation. Third, with a geriatric cohort, the competing risk of hospitalisation and mortality from ageing itself maybe so significant that more appropriate endpoints may be on functional capacity and patient-reported quality of life scores. Another important limitation is the absence of information on the effects of fluid retention, blood

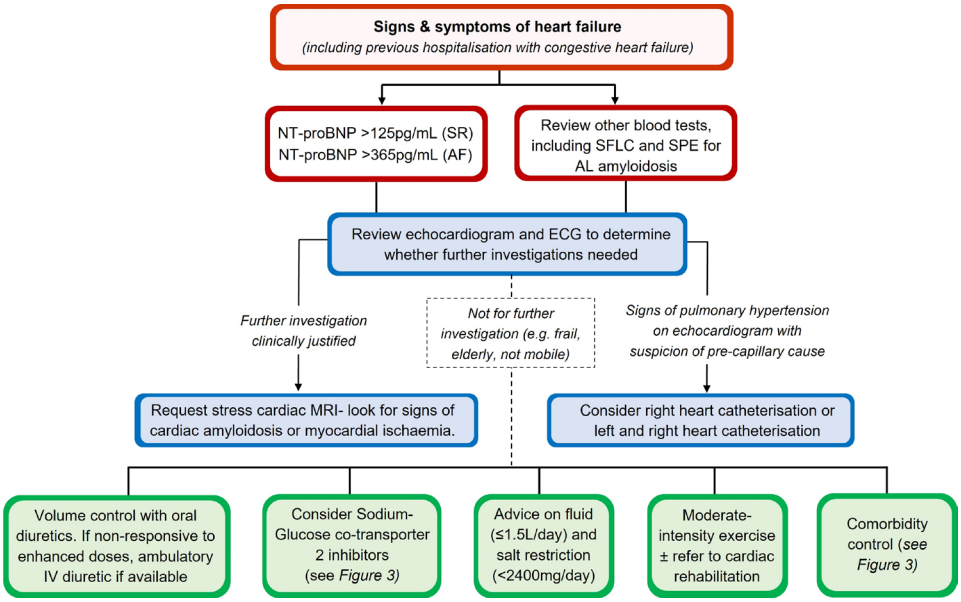

Figure 4 Simplified approach to HFpEF in the outpatient setting, complemented by elements of figure 3. AF, atrial fibrillation; HFpEF, heart failure with preserved ejection; SFLC, serum free light chains; SPE, serum protein electrophoresis; SR, sinus rhythm.

pressure and heart rate after the baseline visit at 6 and 12 months. As previously described, most patients were not routinely followed up and the GP was given the responsibility of getting these parameters to the target level recommended in clinic. Due to this reason and the retrospective study design, we were unable to obtain an adequate number of patients with valid 'before' and 'after' parameters (including blood pressure, weight and NT-pro-BNP) for an accurate paired t-test. A prospective study would therefore be valuable to address this gap. Nonetheless, the neutral outcomes were able to stimulate a discussion and design of a novel pathway that is worthy of consideration in clinical practice and policy-making.

CONCLUSION

With a growing arsenal of diagnostic and therapeutic tools to tackle the prevailing syndrome of HFpEF, an efficient and comprehensive clinic model is needed. However, given the current resource constraints within the NHS, this can be challenging to accomplish. This timely study has demonstrated that existing general and emerging HFpEF clinics may not comprehensively address the multifaceted aspects of HFpEF as clinic activities concentrated primarily on cardiological measures. Hence, aligning with multiple national initiatives, we have proposed a pragmatic and feasible outpatient framework that can facilitate the appropriate prescription of SGLT2i and other future evidenced-based medications, promote a structured approach to assessing HFpEF while avoiding overinvestigations in the frail, maximise the potential of the allied healthcare workforce, and place non-cardiac comorbidities at the forefront of our future dedicated HFpEF clinic service.

Twitter Prithwish Banerjee @banerjeep

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ORCID iDs
Patrick Tran http://orcid.org/0000-0001-9304-9521
Prithwish Banerjee http://orcid.org/0000-0001-7793-1733

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