were extracted from the CHEMOCARE database which allowed us to identify whether cardiac assessments were carried out at baseline and during treatment as per recommendations. LVEF at baseline and during routine follow-up was also calculated to determine if patients had developed left ventricular systolic dysfunction (LVSD) during chemotherapy.

Results Of 1262 patients, 1260 patients (99.8%) were female, with an mean age of 59.3 years. 82.2% of the patients had received anthracyclines alone and 17.8% received a combination of an anthracycline and trastuzumab. 900 patients (71.3%) received adjuvant radiotherapy, 311 (24.6%) received chemotherapy only and 51 (4.0%) had an unknown type of therapy. 1101 (87.2%) of patients were on epirubicin with an average cumulative dose of 570.7 mg/m², making it the most used anthracycline as it is less cardiotoxic compared to doxorubicin. 683 (54.0%) underwent screening for baseline cardiac function using MUGA scans. Of these, only 290 (42.5%) had follow up scans during the course of their treatment as mandated by guidelines. Among patients who had adequate follow up, 32 (11%) developed LVSD during chemotherapy, corresponding to a rate of 6.7% (n = 5) among those on anthracyclines alone and 12.5% (n = 27) among patients on combination therapy.

Conclusion Despite recommendations to monitor cardiac function, our data shows in all chemotherapy patients, only half of them were monitored in our cohort. Although LVSD is usually a late sequel of cancer treatment, we have shown its occurrence even when chemotherapy is being delivered, thus highlighting the importance of surveillance to ensure early detection and management. This may reflect physicians'

opinion, contrary to guideline recommendations, that single chemotherapy is associated with less cardiotoxicity.

24 AN EPIDEMIC OF HFPEF?

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Introduction There is debate about the diagnostic profile of the heart failure population. Diagnostic criteria for heart failure with reduced ejection fraction (HFREF) are variable and many believe that heart failure with preserved ejection fraction (HFPEF) is over-diagnosed. There is also differing opinion about whether these conditions are distinct entities. The aim of this study was to differentiate a heart failure population with contemporary diagnostic parameters.

Methods A diagnostic pathway using current best evidence for the diagnosis of HFREF and HFPEF was created. This was applied prospectively to all 319 new patients attending our heart failure clinic between May 2013 and July 2014.

Results 245 of the 319 new patients were deemed to have heart failure clinically. 73% met our diagnostic criteria for HFREF (LVEF <55%), and only 14% met the diagnostic criteria for HFPEF. Changing the LVEF threshold to <40% produced a ratio of HFREF to HFPEF of 1.3:1 which is more in keeping with current perceptions about an epidemic of HFPEF, but reflects the fact that many of those diagnosed

Abstract 24 Figure 1 HFREF, Heart failure with reduced ejection fraction; HFPEF, Heart failure with preserved ejection fraction; HFNMDS, Heart failure with no major structural disease; LVEF, Left ventricular ejection fraction

Abstract 24 Table 1 Breakdown of heart failure population according to a contemporary diagnostic framework and utilising different LVEF thresholds for HFREF

LVEF threshold for HFREF		Total	HFREF	HFPEF	HFNMDS	HF Alt cause	RHF	HFPEF and HFNMDS	Ratio of HFREF to HFPEF
LVEF <55%	Number	245	180	35	14	13	3	49	5.2 : 1
	% of total	100%	73%	14%	5.7%	5.3%	1.2%	20%	
LVEF <50%	Number	245	163	52	14	13	3	66	3.2 : 1
	% of total	100%	67%	21%	5.7%	5.3%	1.2%	27%	
LVEF <45%	Number	245	141	74	14	13	3	88	1.9 : 1
	% of total	100%	58%	30%	5.7%	5.3%	1.2%	36%	
LVEF <40%	Number	245	123	92	14	13	3	106	1.3 : 1
	% of total	100%	50%	38%	5.7%	5.3%	1.2%	43%	

LVEF, Left ventricular ejection fraction; HFREF, Heart failure with reduced ejection fraction; HFPEF, Heart failure with preserved ejection fraction; HFNMDS, Heart failure with no major structural disease; HF Alt cause, Heart failure due to an alternative cause; RHF, Right heart failure.

with HFPEF have a definite reduction in LVEF that could account for their presentation. A requirement for elevated LV diastolic pressures or elevated biomarkers +/-diastolic dysfunction to justify a diagnosis of HFPEF resulted in a number of individuals being reclassified as having no major structural heart disease. Subset analysis of the 25 patients that maintained a diagnosis of HFPEF following a more detailed echo and MRI showed that 76% had convincing markers of LV systolic dysfunction despite a normal LVEF.

Conclusions Variable diagnostic boundaries mean some definitions of HFPEF incorporate more people with subtly reduced LVEF than others. A set of positive criteria for the diagnosis of HFPEF remains challenging and resting echocardiographic measurements have significant limitations. Currently, no universal diagnostic pathway has been agreed but our framework and analysis help better understand the HFPEF phenomenon.

25 IN HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFPEF), CARDIOVASCULAR MAGNETIC RESONANCE IMAGING (CMR) DETECTS NEW, ALTERNATIVE DIAGNOSES WHICH CARRY PROGNOSTIC SIGNIFICANCE

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Introduction Heart failure with preserved ejection fraction (HFPEF) carries poor prognosis and definitive therapies are lacking. Transthoracic echocardiography (TTE) remains the primary diagnostic modality in HFPEF. We aimed to evaluate the diagnostic and prognostic utility of cardiovascular magnetic resonance (CMR) in HFPEF.

Methods Patients were recruited as part of Developing Imaging And plasMa biOMarkers iN Describing-HFPEF (DIAMOND-HFPEF): a prospective, single-centre study. Inclusion criteria were: clinical or radiographic evidence of heart failure (HF) and ejection fraction > 50% on TTE. Exclusion criteria were: myocardial infarction (MI) in the preceding 6 months, suspected or confirmed cardiomyopathy, constrictive

pericarditis, non-cardiovascular life expectancy < 6 months and severe valve/ lung/ renal disease. The CMR protocol included cine, adenosine stress/rest perfusion and late gadolinium enhancement imaging on a 3-Tesla scanner. Both TTE and CMR were performed during the index study visit and reported independently. Follow-up outcome data was collected at a minimum of 6 months post-enrollment for the primary endpoint (death and/or re-hospitalisation with HF).

Results 154 patients (mean age 72.4 ± 10.0 years, 51% Male) underwent both CMR and TTE. CMR detected the following previously unknown diagnoses (total n = 42): significant coronary artery disease (n = 16, including 14 with 'silent'), microvascular dysfunction (n = 11), hypertrophic cardiomyopathy (n = 10) and constrictive pericarditis (n = 5). During follow-up (median = 623 days, interquartile range 455 – 753), there were 53 primary outcome events. Kaplan-Meier survival analysis (see Figure 1) revealed worse outcomes in the 'new diagnoses group' (Log Rank test p = 0.046). In a multivariate Cox Regression model comprising significant independent predictors during univariate analysis (diastolic blood pressure, NYHA class, urea, eGFR and log BNP), the 'new diagnoses

Abstract 25 Figure 1