Abstract 25 Figure 2

group' (hazard ratio [HR]: 1.917; 95% confidence interval [CI]: 1.064 to 3.454; $p = 0.03$) remained a significant independent predictor of primary outcomes (see Figure 2).

Conclusion In HFPEF, CMR identifies previously unknown pathologies in a significant minority (>25%). This group of 'new diagnoses' is associated with worse outcomes and is an independent predictor of death and/or re-hospitalisation with HF.

Interventional Cardiology

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EFFECT OF AGE AND GENDER ON OUTCOMES FOLLOWING PRIMARY PERCUTANEOUS CORONARY INTERVENTION FOR ST-ELEVATION MYOCARDIAL INFARCTION IN A UK TERTIARY CENTRE

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Abstract 25 Table 1

	Univariable, HR (95% CI)	p Value	Multivariable, HR (95% CI)	p Value
Age	1.014 (0.985–1.045)	0.336		
Gender	1.480 (0.843–2.598)	0.172		
Systolic Blood Pressure	0.995 (0.983–1.007)	0.377		
Diastolic Blood Pressure	0.973 (0.950–0.997)	0.026	0.991 (0.967–1.016)	0.484
NYHA III/IV	1.802 (1.024–3.171)	0.041	1.552 (0.833–2.893)	0.166
Urea	1.086 (1.022–1.153)	0.007	1.104 (1.007–1.211)	0.035
eGFR	0.987 (0.973–1.001)	0.069	1.009 (0.989–1.030)	0.369
ZLog BNP pmol/l	1.471 (1.079–2.006)	0.015	1.442 (1.032–2.015)	0.032
New Diagnoses	1.752 (0.999–3.071)	0.050	1.917 (1.064–3.454)	0.030
Group				

Introduction Techniques and pharmacotherapy for Primary Percutaneous Coronary Intervention (PPCI) continue to evolve rapidly. The West Yorkshire PPCI outcome study was established to identify important clinical and procedural variables that may impact on patient outcomes following PPCI for ST-Elevation Myocardial Infarction (STEMI). We sought to clarify the influence of age and gender on major adverse cardiovascular events (MACE), which have been variably reported, in a large consecutive patient series.

Methods Retrospective analysis of 3049 consecutive patients who underwent PPCI for STEMI between 1-1-2009 and 31-12-2011 at Leeds General Infirmary. Minimum 12m clinical follow-up data were collected for all; MACE was defined as total mortality, myocardial infarction and unplanned revascularisation. Unadjusted and adjusted analyses for total mortality and MACE were performed with Cox proportional hazards models for male ($n = 2223$) and female ($n = 826$) patients and for three age tertiles - under 60yrs (Group 1, $n = 1276$), 60 to 79yrs (Group 2, $n = 1391$) and 80yrs and above (Group 3, $n = 382$).

Results Females (mean 68 ± 13 yrs) had significantly higher rates of total mortality and MACE compared to males (mean 61 ± 13 yrs) (Table 1, Figure 1). However, when adjusted for age, there was no statistically significant difference in total

Abstract 26 Figure 1 Kaplan-Meier survival curves for total mortality and MACE in females and males

Abstract 26 Figure 2 Kaplan-Meier survival curves comparing age-adjusted mortality and MACE in females and males

mortality or MACE between genders (Table 2, Figure 2). Age directly correlated with total mortality and MACE. Comparison between the three age tertiles showed Group 2 was associated with a higher rate of death (HR 4.17, 95% CI 2.86–6.09) and MACE (HR 2.03, 95% CI 1.60–2.57) at 12m compared to Group 1. The highest rate of death (HR 10.53, 95% CI 7.07–15.67) and MACE (HR 3.93, 95% CI 2.99–5.17) was seen in Group 3.

Conclusion This large retrospective study has shown that whilst women appear to have significantly higher rates of death and MACE compared to men, when adjusted for age, gender was not associated with a statistically significant difference in outcomes. The difference in unadjusted outcomes may be due to the fact that women tend to present with STEMI at an older age than men. Therefore, the difference in outcomes is likely to be age-related rather than gender-related.

Abstract 26 Table 1 Total mortality and MACE rates in females and males

Gender	Number n	Death n (%)	MACE n (%)
Male	2223	175 (7.9)	276 (12.4)
Female	826	94 (11.4)	140 (16.9)
		p < 0.01	p < 0.01
		HR 1.48 (1.15–1.90)	HR 1.40 (1.14–1.72)

Abstract 26 Table 2 Age-adjusted mortality and MACE rates in females and males

Gender and Age	Number n	MACE n (%)	Death n (%)
Male <60	1060	78 (7.4)	26 (2.5)
Female <60	216	22 (10.2)	7 (3.2)
		p = 0.16	p = 0.51
		HR 1.40 (0.87–2.25)	HR 1.32 (0.58–3.06)
Male 60–79	976	146 (15.0)	103 (10.6)
Female 60–79	415	65 (15.7)	41 (9.9)
		p = 0.99	p = 0.53
		HR 1.00 (0.75–1.35)	HR 0.89 (0.62–1.28)
Male ≥80	187	52 (27.8)	46 (24.6)
Female ≥80	195	53 (27.2)	46 (23.6)
		p = 0.90	p = 0.83
		HR 0.98 (0.67–1.43)	HR 0.96 (0.64–1.44)

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IMPACT OF CULPRIT VERSUS NON-CULPRIT ANGIOGRAPHY STRATEGY ON PRIMARY PCI DOOR TO BALLOON TIMES

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Background Guidelines for ST-segment elevation myocardial infarction (STEMI) recommend primary percutaneous coronary intervention (PPCI) within 90 min of arrival in a PCI-capable hospital. Some PCI operators perform diagnostic angiography of the non-culprit artery prior to intervention ('non-culprit' strategy) while others proceed directly to the presumed culprit vessel ('culprit' strategy) reserving imaging of the non-culprit vessel until after the PCI. We evaluated the 'time cost' of each approach and their impact upon door to balloon times (D2B).

Methods All consecutive patients presenting with STEMI to a regional heart attack centre between April 2014 and March 2015 (n = 630) were included. The time from the first angiogram acquisition (culprit or non-culprit vessel) to device use (thrombectomy catheter, balloon or stent) was recorded for each strategy. Overall D2B times were analysed.

Results A culprit strategy was followed in 69/630 and a non-culprit approach in 561/630. The mean time from first image to device use was 15 mins 41s for non-culprit strategy and 8 mins 9s for culprit strategy. The non-culprit strategy therefore incurred a delay of 7 mins 32s (p < 0.01). The mean D2B time was 52 mins and 66 mins for the culprit and non-culprit strategies respectively (p < 0.05). The percentage meeting D2B time <90mins was 86% for culprit and 78% for non-culprit strategies. Percentage meeting D2B time <60 mins (which may have additional mortality benefit) was 71% and 58% for the culprit and non-culprit strategies respectively. In our cohort, mortality was significantly lower in patients with D2B <90 mins at 2.0%, versus 4.6% in those whose D2B was >90mins (p = 0.02).

Conclusion A 'culprit' PPCI strategy results in significantly shorter D2B times, facilitating institutional attainment of national guideline targets, which may translate into improved patient outcomes.