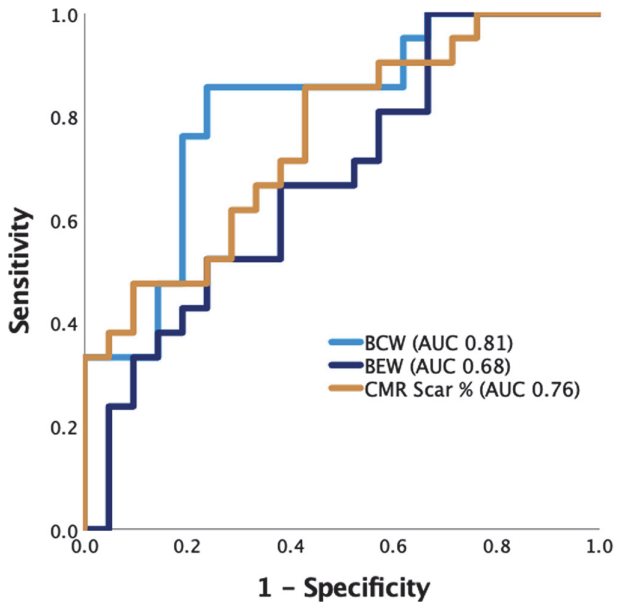
functional recovery. Resting backward compression wave energy was significantly greater in recovering than non-recovering territories (-5240 ± 3772 vs. -1873 ± 1605 W.m-2.s-1, p = 0.099, figure 1), and had comparable diagnostic accuracy to CMR (area under the curve 0.812 vs. 0.757, p = 0.649, figure 2); a threshold of -2500 W.m2.s-1 had 86% sensitivity and 76% specificity at predicting recovery. Backward expansion wave energy did not predict recovery. FFR was numerically higher in recovering territories (0.81 ± 0.17 vs. 0.71 ± 0.16, p = 0.058), whilst hyperaemic microvascular resistance did not differentiate recovering from non-recovering territories (1.97 ± 0.73 vs. 2.29 ± 1.00, p = 0.287). The likelihood of functional recovery was similar in revascularised and non-revascularised territories (15/29 vs. 6/13 respectively, p = 0.739). Low-dose dobutamine stress increased the energy of all waves, but did not improve the accuracy of cWIA in predicting recovery. In a regression model, resting backward compression wave energy and optimisation of medical therapy predicted functional recovery; fractional flow reserve and revascularisation with PCI did not.



Abstract 38 Figure 2

Abstract 38 Table 1 Participant characteristics. No statistically significant differences were observed between the initial population and the subset who had echocardiographic follow up

Demographic	*All participants (N = 40)*	*Participants with follow-up (N = 30)*
Age (years)	66.0 ± 11.2	65.6 ± 11.7
Male (%)	30 (75)	21 (70)
Hypertension (%)	23 (58)	16 (53)
Hypercholesterolemia (%)	29 (73)	21 (70)
Current/prior smoking (%)	23 (58)	17 (57)
Diabetes mellitus (%)	21 (53)	18 (60)
Heart rate (bpm)	74 ± 13	75 ± 12
Systolic blood pressure (mmHg)	127 ± 28	130 ± 28
Diastolic blood pressure (mmHg)	67 ± 14	68 ± 13
Angiography		
Number of diseased vessels	2.37 ± 0.66	2.33 ± 0.65
Number of CTOs	0.61 ± 0.63	0.63 ± 0.66
BCIS Jeopardy Score	10 [8-12]	10 [8-12]
CMR		
LVEDVi (ml/m^2)	124 ± 33	123 ± 34.8
LVESVi (ml/m^2)	91 ± 33	89 ± 34
LVSVi (ml/m^2)	34 ± 10	34 ± 9.8
LVEF (%)	28.7 ± 9.0	29.7 ± 8.4
LV mass index (g/m^2)	66 ± 17	66 ± 18
Scar burden (%)	7.9 [1.5-30.4]	18.7 [11.0-28.1]
RVEF (%)	48.0 ± 11.7	48 ± 12.8
Other		
NT-pro-BNP (pg/ml)	2011 [517-3793]	3306 [632-7023]
hs-cTnT (ng/ml)	38 [19.5-59.5]	38 [13-58]

Conclusions Backward compression wave energy has similar accuracy to late gadolinium enhanced CMR in the prediction of functional recovery. cWIA has the potential to revolutionise the management of ischaemic left ventricular dysfunction, in a manner analogous to the effect of fractional flow reserve on the management of stable angina.

Conflict of Interest None

39 DRUG COATED BALLOON ONLY ANGIOPLASTY FOR STABLE ANGINA IN ROUTINE CLINICAL PRACTICE

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Introduction The recent BASKETSMALL2 trial demonstrated safety and efficacy of drug coated balloon (DCB) angioplasty for de novo small vessel disease. Registry data have demonstrated that DCB angioplasty is safe; however, the majority of these studies are limited due to long recruitment time and small number of patients with DCB compared to drug eluting stents (DES). Our aim was to investigate if DCB-only strategy is safe to incorporate in routine clinical practice.

Methods We identified all patients treated for stable angina and de novo disease in our institution from January 2015 till November 2019. During that period an equivalent number of patients were treated with DCB-only or DES-only strategy on a yearly basis.The primary endpoint was all cause mortality. The secondary endpoints were cardiovascular mortality, acute coronary syndrome (ACS), ischaemic stroke, major bleeding and target lesion revascularisation (TLR). Data were obtained from the hospital episodes statistics from NHS digital. Clinical and angiographic data were collected from our prospectively collated database and supplemented from electronic records as required. All angiograms were reviewed to confirm accuracy of angiographic data and determine TLR.

Results A total of 1302 patients were identified. HES data were not obtained for 65 patients who had opted-out, therefore 1237 were included in the analysis; 544 were treated with DCB and 693 with DES. The average age for the DCB-group was 67.9±10.2 years old (79% male); while for the DES group it was 67.9±9.7 years old (78.1% male). The average follow up was 1339±514 days and 1354±527 days for the DCB and DES group respectively. Table 1 shows the

differences between the groups in terms of clinical and angiographic characteristics.The all cause mortality was 35 (6.4%) and 59 (8.5%) for the DCB and DES group respectively. Kaplan Meier estimator plot did not show a significant difference between the groups. There was no difference between the groups in any of the secondary endpoints (cardiovascular mortality, ACS, stroke, major bleeding and TLR). On multivariable COX regression analysis (Table 2) age, frailty and hypercholesterolaemia were the only independent predictors of mortality.

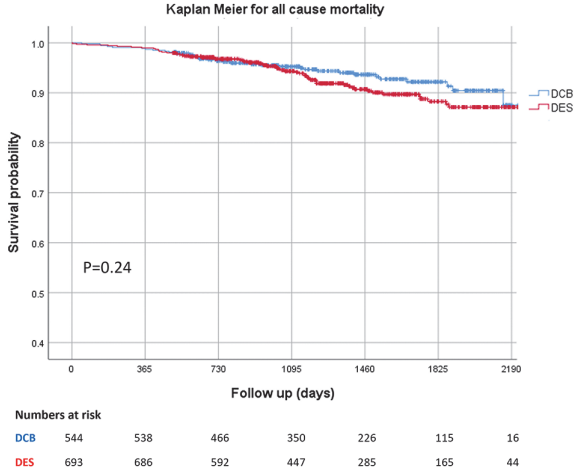
Conclusion Our real world data from a large, contemporary cohort of stable angina patients, including predominantly large vessels, demonstrate that DCB only angioplasty is safe compared to DES in terms of all major cardiovascular endpoints including TLR.

Abstract 39 Table 1 Clinical and angiographic differences between the groups

	DCB (544 patients) 640 lesions	DES (693 patients) 831 lesions	P value
COPD	3%	6.3%	0.01
Vessel diameter	3.09 ±0.53mm	3.46±0.58mm	<0.001
Patients with vessel ≥3mm	398 (73.2%)	594 (85.7%)	<0.001
Patients with true bifurcation	63 (11.6%)	56 (8.1%)	0.04
TIMI flow pre-PCI 0 or 1	75 (11.7%)	55 (6.6%)	0.001

Abstract 39 Table 2 Multivariable cox regression analysis for all-cause mortality

	Hazard ration (95% CI)	P value
Age	1.075 [1.046-1.105]	<0.001
Hypercholesterolaemia	0.579 [0.339-0.989]	0.046
Frailty	1.334 [1.197-1.486]	<0.001



Abstract 39 Figure 1

Conflict of Interest N/A

40 IMPACT OF TIME FROM SYMPTOM ONSET ON THE DIAGNOSTIC PERFORMANCE OF HIGH-SENSITIVITY CARDIAC TROPONIN FOR TYPE 1 MYOCARDIAL INFARCTION

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Introduction In patients with suspected acute coronary syndrome, cardiac troponin is used to identify those at high and low risk of future cardiovascular events. The ability of high-sensitivity assays to quantify low levels of cardiac troponin has led to the development of algorithms which allow the early rule-out or rule-in of myocardial infarction on the basis of a single presentation sample. The performance of a single test approach will in part be dependent on the time taken for troponin to reach a specified risk stratification threshold following injury. Guidelines recommend single test strategies are not used in early presenters. However, it is unclear how diagnostic performance changes over multiple time points and whether caution should be applied when using single test strategies in late presenters.

Methods In a secondary analysis of a multicentre trial of consecutive patients with suspected acute coronary syndrome, we evaluated the diagnostic performance of presentation high-sensitivity cardiac troponin I to rule-out or rule-in type 1 myocardial infarction across four groups based on the time from symptom onset to troponin sampling (<3 hours, 4 to 6 hours, 7 to 12 hours and ≥12 hours) using common rule-out (<2ng/L & <5ng/L) and rule-in (>99th centile, >64ng/L) thresholds.

Results In 41,104 (mean 60 (49–74) years), 3,692 (9%) patients had an adjudicated diagnosis of type 1 myocardial infarction. For both rule-out thresholds, sensitivity for was highest 7–12 hrs following symptom onset and lowest in patients presenting within 3 hrs. In patients presenting within 3 hrs, a rule-out threshold of <2ng/L resulted in superior sensitivity and negative predictive value compared with a threshold of 5ng/L (99.4% [95% confidence intervals 98.9–99.7%] and 99.7% [99.5- 99.9%] vs 96.7% [95.7- 97.6%] and 99.3% [99.1 to 99.5%] respectively). For the rule-in of myocardial infarction, sensitivity for the 99th centile and >64ng/L threshold was lowest in patients presenting within 3 hrs (71.7% [69.3–74.1%] vs 46.5% [44.1–49.2%] respectively), increased with time from symptom onset and was highest in those presenting >12 hrs from symptom onset (92.5% [90.5–94.5%] vs 70.0% [66.8–73.2%] respectively). Specificity and positive predictive value for both thresholds was greatest 7–12 hrs following symptoms and declined in those presenting >12 hrs after symptom onset.

Conclusion The ability of a single cardiac troponin sample to rule-in or rule-out type 1 myocardial infarction is influenced by the time from symptom onset to troponin sampling with. A limit of detection strategy may facilitate the safe rule-out of type 1 myocardial infarction in patients presenting within 3 hrs from symptom onset.

Conflict of Interest None