gender, education level, history of angina, history of myocardial infarction, history of angioplasty, previous coronary artery bypass surgery, diabetes, current smoking status, hypercholesterolaemia and hypertension. Those who perceived themselves to be at higher risk of a future event were more likely to be female (p<0.001), have a higher education level (p=0.02), aged between 44 and 59 (p=0.02) and have a previous history of percutaneous coronary angioplasty (p=0.003).

Conclusions/Implications Despite the fact that the entire study sample were diagnosed with ACS, approximately one in five (20%) perceived that they were less likely than other people of the same age to experience a heart attack in the future. These findings highlight the need for specific and targeted education of both men and women in Ireland with respect to the risks for heart disease and heart attacks.

| 124 | IMPACT OF CONTRAST-INDUCED NEPHROPATHY UPON SHORT AND LONG-TERM OUTCOMES OF PATIENTS WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION UNDERGOING PRIMARY PERCUTANEOUS CORONARY INTERVENTION |

Sean Gallagher,* S Hassan, D A Jones, M J Lovell, A Ahktar, A Kapur, C Knight, A Mathur, M M Yaqoob, A Wragg. *Barts and the London NHS Trust, London, UK*

Introduction Contrast induced nephropathy (CIN) is associated with adverse clinical outcomes, including prolonged hospitalisation and increased morbidity and mortality following elective invasive cardiac procedures. The impact of CIN following primary PCI (PPCI) for ST segment elevation MI (STEMI) remains poorly defined.

**Aim** To investigate the long-term prognostic implications of CIN following PPCI for STEMI.

Methods This is a retrospective observational registry study. Data were available upon 2224 patients undergoing PPCI for STEMI at a tertiary Cardiac centre between October 2003 and May 2010. CIN was defined as an increase in serum creatinine (>25% or 44.2 mmol/l) within 2 days of PPCI. The primary outcome measure was all-cause mortality determined via Office of National Statistics data.

Results CIN was observed in 317 patients (14.3%). Patients with CIN were older (69.3 vs 62.7, p<0.0001), more likely to be female (30.0% vs 22.6% p=0.004), had more vascular risk factors (including diabetes, hypertension and chronic kidney disease), had more previous MIs (17.7% vs 11.7%, p=0.003), more multivessel CAD

(55.2% vs 40.6%, p<0.0001), and more LV dysfunction (52.1% vs 32.1%, p<0.0001) (Abstract 124 table 1). Length of hospital stay (5 vs 2 days, p<0.0001), 30-day mortality (16.4% vs 2.0%, p<0.0001) and 3-year mortality (22.4% vs 5.0%, Log rank p<0.0001) (Abstract 124 figure 1) was worse in patients with CIN. CIN was an important independent predictor of all cause mortality (HR 2.71, CI 1.97 to 3.75). Other independent predictors of mortality included age>75 years (HR 1.81, CI 1.28 to 2.57), eGFR <60 mls/min (HR 2.36, CI 1.69 to 3.31), cardiogenic shock (HR 3.98, CI 2.76 to 5.73), LV dysfunction (HR 1.82, CI 1.34 to 2.48) and multivessel CAD (HR 1.59, CI 1.15 to 2.20).
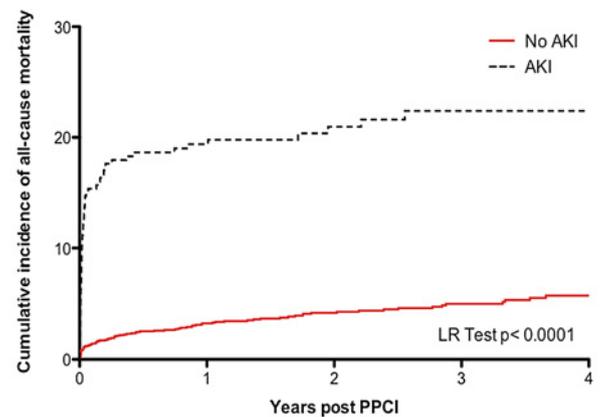
Abstract 124 Figure 1 Effect of AKI upon all-cause mortality following PPCI for STEMI.

Conclusion CIN following PPCI for STEMI was associated with increased short and long-term mortality and increased length of hospital stay. Better strategies are needed to prevent CIN in high risk STEMI patients.

| 125 | MONOCYTE SUBPOPULATION COUNTS AND FUNCTIONAL CHARACTERISTICS PREDICT ADVERSE CLINICAL EVENTS POST ST ELEVATION MYOCARDIAL INFARCTION |

[1]A A Ghattas,* [1]G Y H Lip, [2]H Griffiths, [2]E Shantsila. [1]*Centre for Cardiovascular Sciences, University of Birmingham, Birmingham, UK;* [2]*Schoolf of Life and Health Sciences Aston University, Birmingham, UK*

Background Monocytes are implicated in the initiation of the atherosclerotic plaque through to plaque instability and rupture during presentation with an acute coronary syndrome (ACS). Little is known about the predictive role of monocytes on clinical outcome. We studied the role of the three phenotypically and functionally discrete monocyte subpopulations in predicting major adverse cardiac events (MACE)—defined as recurrent ACS, heart failure and death- following ST elevation myocardial infarction (STEMI).

Method STEMI patients treated with percutaneous revascularisation, were recruited in the first 24 h post-infarction. Peripheral blood monocyte subpopulations were enumerated and characterised using flow cytometry after staining for CD14, CD16 and CCR2. Phenotypically, monocyte subpopulations are defined as: CD14++CD16−CCR2+ (Mon1), CD14++CD16+CCR+ (Mon2) and CD14+CD16++CCR2− (Mon3) cells. Functionally, monocyte subpopulation activation of nuclear factor κ B (NFκB) was analysed. Activation of NFκB was determined by flow cytometry as the mean

Abstract 124 Table 1 Clinical characteristics of study cohort

	No AKI (n=1907)	AKI (n=317)	Significance
Age (yrs±SD)	62.7±13.8	69.3±1.8	0.004
Age>75, n (%)	404 (21.2)	128 (40.4)	<0.0001
Female, n (%)	431 (22.6)	95 (30.0)	<0.0001
Hypertension, n (%)	720 (37.8)	147 (46.4)	0.004
Diabetes mellitus, n (%)	324 (17.0)	81 (25.6)	<0.0001
Hypercholestrolaemia, n (%)	1120 (58.7)	149 (47.0)	<0.0001
Smoking history, n (%)	554 (29.1)	98 (30.9)	0.500
eGFR <60 mls/min, n (%)	355 (18.6)	105 (33.1)	<0.0001
Mean eGFR (±SD)	77.6±21.0	75.1±31.4	0.06
Previous MI, n (%)	223 (11.7)	56 (17.7)	0.003
Previous CABG, n (%)	52 (2.7)	12 (3.8)	0.296
Previous PCI, n (%)	180 (9.4)	36 (11.4)	0.286
EF <50%	613 (32.1)	165 (42.1)	<0.0001
Multivessel CAD, n (%)	774 (40.6)	175 (55.2)	<0.0001
Cardiogenic Shock, n (%)	88 (4.6)	43 (13.6)	<0.0001

fluorescent intensity (MFI) of intracellular κ-B kinase β (IKKβ), as a downstream activation product of the NFkB pathway. MACE events were recorded at follow-up.

Results We recruited 96 patients (average age 61.5 years±13.3; 64.6% male). Patients were followed-up for a median of 187 days (112−222 days). MACE events occurred in 14 patients (14.6%). Using logistic regression analysis, increased total monocyte count ($p < 0.032$), Mon2 counts ($p < 0.047$) and Mon3 IKKβ ($p < 0.013$) were significantly predictive of MACE at follow-up (Abstract 125 table 1). Mon2 counts were an independent predictor of MACE after adjusting for age and sex.

Abstract 125 Table 1 Monocyte subpopulations and IKKβ predict MACE

	Monocytes	OR (95% CIs)	p Value
Phenotypic characterisation and enumeration	Total Mon	1.002 (1 to 1.004)	0.032
	Mon1	1.001 (0.998 to 1.003)	0.111
	Mon2	1.008 (1.003 to 1.013)	0.047
	Mon3	1.01 (0.999 to 1.022)	0.388
Functional assessment	IKKβ Mon1	0.982 (0.944 to 1.022)	0.388
	IKKβ Mon2	0.983 (0.948 to 1.019)	0.373
	IKKβ Mon3	1.038 (0.993 to 1.086)	0.013

Conclusion Increased total monocyte and Mon2 counts in the first 24 h post infarction is predictive of MACE in STEMI patients. Mon3, despite an assumed role in reparation and fibroblast deposition, are also predictive of MACE. Monocytes remained functionally active throughout the acute and healing phases, and thus may have prognostic implications.

126	**DYNAMICS OF THE THREE HUMAN MONOCYTE SUBSETS OVER 30 DAYS IN ST-ELEVATION MYOCARDIAL INFARCTION**

L D Tapp,* B J Wrigley, B Pamukcu, E Shantsila, G Y H Lip. *City Hospital, Birmingham, UK*

Introduction Monocytes are intimately involved in the pathophysiology of myocardial infarction (MI), with different subsets thought to have distinct roles in cardiac repair. We have described that human monocytes can be divided phenotypically into 3 subsets by their surface expression of CD14, CD16 and CCR2: CD14 +CD16−CCR2+ (Mon1, "classical" monocytes), CD14+CD16 +CCR2+ (Mon2) and CD14lowCD16+CCR2− (Mon3). Having observed a threefold increase in Mon2 at admission with STEMI, we aimed to establish the dynamics in total monocyte count, subset count and relative proportions of the subsets in the 30 days following ST elevation myocardial infarction (STEMI) treated by primary percutanous coronary intervention (PPCI).

Methods Monocyte subsets were measured by flow cytometry in 50 patients (57.5±11.7 years, 86% male) with STEMI at 4 time points: within 24 h after PPCI, day 3, day 7 and day 30 after MI onset. All patients underwent PPCI. Exclusion criteria comprised factors known to affect monocyte count.

Results The peak number of total monocytes, Mon1 and Mon2 occurred on day 1, with comparable values on day 3. The total monocyte count and Mon1 reduced significantly by day 30, to levels seen is stable coronary artery disease. Mon2 count reduced significantly earlier, by day 7. No changes were seen in Mon3 count. Mon2 predominates over Mon3 on day 1, with the reverse pattern seen at day 30, where the Mon2 proportion had reduced significantly and the Mon3 proportion had increased significantly.

Conclusions We observed prominent differences in the dynamics of monocyte subsets, particularly the minor subsets (Mon2 and Mon3) which have been suggested to play distinct roles in myocardial reparative processes. The dramatic increase in number of Mon2 after STEMI followed by significant reduction by day suggests a specific role in the acute phase of MI. These novel findings may contribute to further understanding the pathophysiology of the recovery processes following acute MI.

Abstract 126 Table 1

	Day 1	Day 3	Day 7	Day 30	ANOVA p value
Total monocytes (per μl)	994±66.2	967±71.0	861±70.3	670±32.4* †	<0.0001
Monocyte subsets					
Mon1	810±57.7	785±58.5	712±62.9	557±31.3* †	<0.0001
Mon2	108±16.0	105±15.9	73.6±13.1†	45.3±5.61* †	<0.0001
Mon3	72.2±7.07	77.1±10.1	75.1±5.71	67.3±4.31	0.55
Relative proportions					
Mon1 %	81.4±1.61	81.3±1.60	82.1±1.55	82.4±1.33	0.81
Mon2 %	10.9±1.36	10.5±1.24	8.58±1.37	7.36±1.05* †	0.002
Mon3 %	7.61±0.82	8.23±0.81	9.37±0.68	10.3±0.64* †	0.001

*$p < 0.05$ vs day 1.
†$p < 0.05$ vs day 3.

127	**DEMONSTRATION OF INTRACORONARY MICROPARTICLE EXPRESSION AND THEIR ASSOCIATION WITH ACTIVATED PLATELET MONOCYTE AGGREGATE IN HUMAN ST ELEVATION MYOCARDIAL INFARCTION**

[1]C A Mavroudis,* [2]D Eleftheriou, [1]B Majumder, [1]R Sapsford, [1]J North, [1]M Lowdell, [2]P Brogan, [1]R D Rakhit. [1]*Royal Free Hospital, London, UK;* [2]*Institute of Child Health, London, UK*

Background Platelets play a central role in the pathophysiology of acute coronary syndrome (ACS). During ACS activated platelets express p-selectin which binds to the p-selectin glycoprotein ligand on the monocyte, forming platelet monocyte aggregates (PMA). PMA expression is a sensitive marker of platelet activation. A microparticle (MP) is a submicron membrane vesicle derived from virtually any cell during various biological processes (cell activation, differentiation or apoptosis). Recent experimental and clinical data point towards a causal effect of MP and mainly platelet derived MP [PMP] in the pathogenesis and disease progression of atherosclerosis and coronary artery disease.

Aim To investigate the relationship between levels of intracoronary MP and PMA in ST elevation ACS.

Methods Patients with ST elevation ACS who underwent primary percutaneous coronary angioplasty were recruited. Blood samples for PMA, MP levels and soluble marker of platelet activation (p-selectin) were collected from the infarct related coronary artery. PMA and MP levels were estimated using fluorescent monoclonal antibodies and flow cytometry. CD61+CD14+CD62P+ events are PMA expressing p-Selectin [activated PMA] and CD61+CD14 +CD142+ events are PMA expressing tissue factor [TF+ PMA]. Total MPs were identified as AnV+ MP and PMP as AnV+CD42 +/or AnV+CD42+CD62+ MP. Endothelial derived MPs (EMP) were identified as AnV+CD105+CD42− events and/or AnV +CD62E+CD42− events. ELISA was used for p-selectin measurement.

Results The mean total AnV+ MP was 3 661 000/ml of plasma. PMP was statistically higher than EMP $p = 0.01$ (mean PMP (SD) 879 986/ml (1 166 000), mean EMP (SD) 89 099/ml (38 867). There was a strongly positive correlation between total Ann V+ MP with