event, more recent studies suggested that a reasonable amount of patients with ISR many develop ACS as the first manifestation of this adverse event. The aim of this study was to determine the different clinical presentations of ISR in a large cohort of consecutive, non-selected patients and compare with native coronary disease.

Methods 14,445 consecutive patients underwent PCI at a single centre (October 2003—May 2010), we identified 922 (6.4%) cases presenting with restenosis after previous PCI. All patients with restenosis presented with new or recurrent symptoms. Demographic and procedural data were collected at the time of intervention (Abstract 36 table 1). In-hospital MACE (myocardial infarction, urgent revascularisation, stroke or death) was documented at discharge. All cause mortality data was obtained from the Office of National Statistics via the BCIS/CCAD national audit out to 3.2 years (mean 3.1 ± 1.3 years).

Abstract 36 Table 1

<table>
<thead>
<tr>
<th>Total</th>
<th>Restenosis n = 922</th>
<th>Native disease n = 13,523</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>63.09</td>
<td>63.76</td>
<td>0.0868</td>
</tr>
<tr>
<td>Ethnicity (cau)</td>
<td>683 (74.2%)</td>
<td>9160 (97.8%)</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>Previous MI</td>
<td>411 (44.6%)</td>
<td>2160 (23.1%)</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>120 (13.0%)</td>
<td>684 (6.9%)</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>DM</td>
<td>299 (32.5%)</td>
<td>1986 (21.2%)</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>HTN</td>
<td>545 (59.1%)</td>
<td>4170 (44.5%)</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>Hichol</td>
<td>544 (59.0%)</td>
<td>3540 (37.8%)</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>Card Shock</td>
<td>6 (0.7%)</td>
<td>100 (1.1%)</td>
<td>0.2339</td>
</tr>
</tbody>
</table>

Results Restenosis presented in 60.4% as stable angina, 30.6% as unstable angina/Non-ST elevation MI and 9% with ST-elevation Myocardial Infarction. Cardiogenic shock was reported in 6 patients (0.6%). Women had a higher incidence of unstable angina/non-STEMI compared with men (52.6% vs 29.1%) but a lower incidence of STEMI (5% vs 10.4%). Baseline characteristics are listed in Abstract 36 table 1. Mortality rate was 0.98% at 30 days, 3.9% at 1 year and 8.7% at 5 years in patients with restenosis. Comparing the restenotic group with those undergoing PCI for de novo coronary artery disease, there were similar ages and incidence of cardiogenic shock but the restenotic group had higher rates of baseline risk factors (diabetes, hypertension, hypercholesterolaemia) and higher rates of previous CABG and MI. There was also a higher proportion of South Asians in the restenotic group. See Abstract 36 table 1. Comparing outcome measures, there were similar rates of inhospital MACE in the 2 groups and over a 5-year follow-up period, there was no difference in all cause mortality. There was no difference in outcome of patients with restenosis vs de novo coronary artery disease regardless of presentation (angina, UA/ NSTEMI/STEMI). See Abstract 36 figures 1 and 2.

Abstract 36 Figure 1 Comparison of mortality between restenosis and no restenosis in STABLE.

Abstract 36 Figure 2 Comparison of mortality between restenosis and no restenosis in ACS.

Conclusions Clinical in-stent restenosis can frequently present as MI and such patients are more likely to have an aggressive angiographic pattern of restenosis. Drug-eluting stents with improved designs or drug elution systems that further decrease the incidence of ISR are needed.

Abstract 36 Figure 3 Decrease in MACE rates associated with drug eluting stent use in patients with diabetes undergoing PCI in large diameter coronary arteries.

Introduction Both large multi centre trials and registry studies have demonstrated that PCI with drug eluting stents (DES) is associated with reduced MACE and restenosis rates compared to bare metal stents (BMS) in native coronary vessels, although this benefit is less evident in those patients with a larger coronary vessel diameter and MACE rates may actually paradoxically increase in this cohort as observed in the BASKET trial. In diabetic patients, a similar or even greater absolute reduction in MACE rates / restenosis risk is seen associated with DES use, although it is unclear as to whether any benefit persists in those with larger diameter native coronary vessels. Previous data derived from diabetic patients in large diameter native coronary vessels has come from registry studies in which numbers were either small (<200 patients) or were from highly selected patient sub groups excluding high-risk individuals (SCAAR registry).

Methods We therefore retrospectively studied 1165 consecutive diabetic patients with target vessel diameter ≥3 mm admitted to our centre for PCI from 2005 to 2009, the largest series of its kind to date. Primary endpoint was defined as total mortality and secondary endpoint was major adverse cardiac event (MACE) defined as composite endpoint of Death, Stroke, MI, Stent Thrombosis and Target Lesion / Vessel Re-Vascularisation.

Results Of the 1165 patients studied, 170 had BMS and 995 had DES. Mean follow-up period was 43.5 ± 21.8 months (median 41.5 months). 73.5% were male in the BMS cohort vs 73.1% in the DES cohort (p > 0.05). Mean age was 62.3 ± 11.2 in BMS and 62.3 ± 10.4 years old in DES (p = 0.55). Other demographic parameters were similar in both groups. There were a total of 23/170 deaths in BMS cohort (13.5%) and 91/995 in DES cohort (9.1%). (HR 1.38; 95% CI 0.83 to 2.27, p = 0.21). A total of 42/170 (24.7%) and 163/995 (16.3%) MACE events were observed in the BMS and DES cohort respectively (HR 1.49; 1.02 to 2.19, p = 0.04). Multivariate analysis
illustrated that use of BMS was independently associated with increased risk of MACE (HR 1.54; 95% CI 1.05 to 2.25, p=0.03), driven through an increase in revascularisation.

Conclusion In conclusion, in one of the largest analyses of its kind, use of DES in patients with diabetes in a real world setting undergoing PCI in large diameter coronary vessels (≥3 mm) is safe and is independently associated with a reduction in MACE events. This is in contrast to that of non-diabetic patients where the benefits of DES in large diameter coronary vessels are less evident.

FALSE ACTIVATION FOR PRIMARY PERCUTANEOUS CORONARY INTERVENTION IS NOT A BENIGN PHENOMENON

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Introduction Primary percutaneous coronary angioplasty (PPCI) is the preferred reperfusion strategy following an acute ST elevation myocardial infarction (STEMI). Since 2005 24/7 primary PCI has been the first line treatment for an acute STEMI in our centre. 95% of patients are direct access admissions by London Ambulance but a significant proportion (up to 20%) do not fulfill the diagnostic criteria for STEMI and are termed “false activations”. Data on the outcome of this cohort of patients is limited.

Aim To review the clinical outcome of patients presenting to our heart attack centre with false activation PPCI.

Method From January 2008 until October 2010, we identified 209 false PPCI activations defined as patients with incomplete diagnostic criteria for acute STEMI: absence of chest pain and/or typical ECG features (ST elevation or new LBBB). Data was collected via a “false activation” database together with retrospective review of case records.

Results Complete data was available in 165 cases. 71% were male and 29% were female (mean age 67). The mean length of stay was 4 days (range 1 – 33). 71% presented with chest pains and 29% had no chest pains, but presented with breathlessness, palpitations or syncope. The ECG abnormality was non-specific ST-T changes in 22%, LBBB in 19%, left ventricular hypertrophy in 15%, fixed ST elevation or Q waves in 14%, early repolarisation changes in 10%, RBBB in 8% and other ECG abnormalities in 12%. The final diagnosis was non-ST elevation acute coronary syndrome (NSTEMACS) in 19%, sepsis in 19% and congestive heart failure (CHF) in 15%. Stable angina was observed in 8% and syncope in 7%. Musculoskeletal or non-cardiac chest pains were noted in 8% and 7% of the patients respectively. 2% of the patients had pulmonary embolism and in 8%, a gastric cause for presentation was diagnosed. 14% had other cardiac problems, including arrhythmia, dilated cardiomyopathy, hypertension, pericarditis, pericardial effusion and late presentation STEMI. 15% had other diagnoses. The mean follow-up period was 18.7 months, during which 21.5% of false PPCI activation admissions died (n=45). 25% (n=11) died during the index admission and 53% (n=15) died within 50 days of admission. The overall 30-day mortality for false activations was 7.2%, which is higher than the overall PPCI mortality of 6.0% (including cardiogenic shock) (p=0.008) and 5.5% (excluding shock) (p<0.0001) in our centre. 49% of deaths were cardiac (NSTEMACS and CHF), 29% sepsis and 22% other causes. The mean age for this cohort was 83.

Conclusion Patients presenting with false PCI activation have a high observed mortality. This is probably due to significant associated comorbidities, including occult cardiac disease. Thus, false PCI activation is not a benign phenomenon and masks underlying significant disease. Robust pathways are required to minimise delay in further investigations and a need for risk stratification for a significant proportion who present with NSTEMACS.

A RANDOMISED CONTROLLED TRIAL COMPARING CONVENTIONAL CORONARY ARTERY BYPASS GRAFT SURGERY WITH A COMPOSITE ARTERIAL GRAFT TECHNIQUE

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Background Composite (Y/T) coronary artery bypass graft surgery (CABG) confers full arterial revascularisation, and “hands off” aorta compared to conventional bypass graft surgery. However, the composite surgical configuration could lead to preferential blood flow down one arm than the other (left internal mammary artery LIMA or radial artery RA) with its potential impact on graft patency.

Aim To investigate the impact of bypass graft configuration on short-term grafts patency and cardiac related quality of life.

Methods and Results This is a single centre randomised, controlled trial Between March 2006 and July 2007, 322 patients undergoing isolated bypass graft surgery at our institution were screened and 89 (27%) met the inclusion criteria and were randomised. Patients were allocated to conventional (conv n=46) or composite (comp n=43). The two primary end points were graft patency defined as (Thrombolysis In Myocardial Infarction) TIMI III flow in distal anastomosis at angiography 12–24 months after surgery; and cardiac-related health status assessed by Seattle angina questionnaire (SAQ). Baseline characteristics were similar between the two groups apart from diabetes where there were more diabetic patients in the composite arm than the conventional one (15(35%) vs 5(11%) p<0.01 respectively). Trial was stopped prematurely following 18 months interim analysis which showed significant graft failure in the composite arm (40%). Final Analysis was performed on intention to treat basis. Sixty-five (73%) had follow-up angiography (34 conv, 31 comp), with total of 116 graft in conventional arm and 100 grafts in composite arm. All patients in both groups had LIMA graft to left anterior descending artery (LAD). Graft patency rate was significantly higher in the conventional compared to composite arm (95(82%) vs 59(59%) p<0.01 respectively). Three main domains of the SAQs that were of significant improvement between before and 6 months after surgery in both groups. There were no significant differences between the two groups in the percentage of improvement in these four domains (Physical limitation, Angina stability, Angina frequency, Quality of life).

Conclusions In our randomised trial, composite bypass graft surgery was associated with higher graft failure rate at 12–24 months after surgery compared to conventional type. This difference may be due to the composite conduit configuration. Further blood flow characteristics study in this configuration can help understand such an important finding and its implication on our clinical practice. Despite the difference in graft patency there were no differences in physical limitation, angina stability, angina frequency, or quality of life between the two groups.

PATIENT VS PHYSICIAN REPORTED ANGINA BEFORE AND AFTER REVASCULARISATION OF CORONARY ARTERY DISEASE: EVIDENCE FROM A LARGE RANDOMISED CONTROLLED TRIAL (THE SOS TRIAL)

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Introduction The success of revascularisation therapies for coronary artery disease (CAD) must be measured by both an improvement in hard clinical endpoints—mortality, repeat revascularisation procedures and myocardial infarction, the traditional focus of clinical trials—and, critically for patients, the relief of angina symptoms. Interest in patient reported outcomes (PROMs) has increased, although their use in cardiovascular trials is far from universal. In