B001

Coronary assessment in patients presenting with stable chest pain: multi-morbidity service evaluation in a large UK based regional cardiac centre


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Objective: Chest pain or discomfort due to angina could have a potentially poor prognosis, emphasising the importance of prompt and accurate diagnosis. National Institute of Clinical Excellence (NICE) of UK has published a\textsuperscript{1} Chest pain of recent onset\textsuperscript{2} guidelines in March 2013. These guidelines appraise the role of latest non-invasive modalities in cardiac imaging to promptly and cost-effectively diagnose coronary artery disease. We aimed to study the service requirement for non-invasive cardiac imaging in patients with stable chest pain using this guidance.

Method: Design: Single centre, six months (January 2010 to June 2010) observational study. Setting: Rapid Access Chest Pain Clinics (RACPC) in a large UK based University Teaching Hospital providing secondary care Cardiology services. Clinical letters were used to ascertain the type of chest pain and cardiovascular risk factors. The resting 12 lead electrocardiogram (ECG) was examined for any ischaemic changes. Patients were then retrospectively allocated to an assessment pathway based on NICE guidance for the evaluation of stable chest pain. Pre-test likelihood of Coronary artery disease (CAD) was calculated using Pryor et al\textsuperscript{3}'s table as published by NICE. Depending on the calculated pre-test probability, their NICE suggested investigation was determined. This included - no further investigations, cardiac computed tomography (CT), functional imaging or invasive angiography

Result: 500 patients were seen in RACP, out of which, 65 patients did not meet the referral criteria of having chest pain. 52% of patients were likely to have Exercise Tolerance Test (ETT) based on previous practice. According to current NICE guidance as applied to our cohort of patients, 128 (30%) would have required functional imaging, 119 (27%) no further investigation, 95 (22%), cardiac CT and 93 (21%) invasive angiography respectively.

Conclusion: Functional imaging and then cardiac CT are the dominant investigations which would be required in the assessment of stable chest pain patients.

B002

Sitagliptin therapy enhanced circulating number of endothelial progenitor cells and angiogenesis: in vitro studies and rat critical limb ischemia model

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Objective: We tested the hypothesis that sitagliptin treatment increased rat critical limb ischemia (CLI) blood flow through enhancing angiogenesis.

Method: Adipocytes were derived from epididymis-adipose tissue of adult-male Fischer 344 rats (n = 6) and cultured in EPG-culture medium with and without sitagliptin (20 \mu M) for 14 days. CLI was performed by ligating rat left femoral artery. Rats (n = 24) were equally divided into group 1 (control), group 2 (CLI only), group 3 (control + sitagliptin [4mg/kg/day]) and group 4 (CLI + sitagliptin). Flow cytometry and Western blot showed EPG (CD34 + , CXCR4 -) and protein expressions (VEGF, CXCR4, SDF-1a) were significantly higher in with than in without sitagliptin treatment at day-14 cell culturing (all p < 0.001), respectively. By day-2 CLI, circulating EPG numbers were similar among groups 1 to 3, but it showed higher in group 4 than in other groups (p < 0.001). Additionally, gene and protein expressions of eNOS, SDF-1a, and VEGF as well as immunofluorescent stains of CXCR4 + , SDF-1a + , VWF + and CD31 + cells in ischemic area were higher in group 4 than in other groups (all p < 0.01). Laser Doppler showed the ratio of ischemic/normal blood flow was higher in groups 1, 3 and 4 than in group 2 (all P < 0.01), but it showed no difference among groups 1 to 3.

Conclusion: Sitagliptin therapy enhanced circulating EPG numbers, angiogenesis and blood flow in CLI setting.

B003

Long-term follow-up evaluation of neointimal coverage and stent apposition after sirolimus-eluting stent implantation by optical coherence tomography


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Objective: Late stent thrombosis related to delayed endothelialisation and incomplete stent appositions are major concerns after drug-eluting stent (DES) implantation. The long-term vascular response towards DES implantation is still unclear. Optical coherence tomography (OCT) is a high resolution modality which provide new opportunities for evaluating the vascular healing reaction after stent implantation.

Method: Fifty two patients who accepted 64 sirolimus-eluting stents (SESs, Cypher Select) were enrolled in the study. The OCT procedure were performed in these 52 patients at 12 months (G1, n = 20), 24 months (G2, n = 17), or 48 months (G3, n = 15) after SESs implantation, respectively. The neointimal hyperplasia (NIH) thickness and stent struts apposition at 1-mm interval and the presence of thrombus in each stent were observed.

Result: The neointimal thickness was significantly higher at 48 months than that of 12 months (0.1694 mm ± 0.1455 mm in G3 vs. 0.1455 mm ± 0.1373 mm in G1, p < 0.01) and 24 months (0.1514 mm ± 0.1296 mm in G2, p < 0.01) after stent implantation, but no significant difference existed between that of 12 months and 24 months (p > 0.05). Longer follow-up duration was associated with significant decrease in the prevalence of uncovered struts (17.26% in G1 vs. 8.76% in G2 vs. 2.56% in G3, p < 0.01) and malapposed struts (14.19% in G1 vs. 10.29% in G2 vs. 4.68% in G3, p < 0.01). The incidence of intrathrombotic presentation steadily decreased trend from 12 months to 48 months (3.82% in G1 vs. 2.41% in G2 vs. 0.81% in G3, p < 0.01).

Conclusion: Neointimal growth continued for as long as 48 months after SES implantation. Neointimal thickness increased non-remarkably from 12 months to 24 months, but significant progress presented at 48 months after stent implantation. Late neointimal growth was accompanied by a higher rate of covered struts and lower rate of malapposed struts.

B004

Atrial electrical and geometrical remodelling at mid-term following device closure of atrial septal defects in adults: a prospective study

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Objective: Late-onset atrial arrhythmia after successful closure of atrial septal defect (ASD) is not uncommon, which may compromise patients’ survival. Right atrial enlargement and electrocardiographic (ECG) p-wave dispersion independently predict the development of atrial arrhythmia. Data are scant to address the degree of right atrial and electrical remodelling and their relations following device closure of ASD.

Method: Echocardiography and ECG were done in 50 consecutive patients (43 ± 17 yrs, 10 males) before and at 3 month after ASD closure. Incomplete right atrial reverse remodelling (RAR) was defined as right atrial volume index (RAVI) ≥21mL/m\textsuperscript{2} at 3 month. P-wave dispersion was calculated as the difference between maximal and minimal p-wave durations in 12-lead ECG.

Result: Before closure, all patients had enlarged right atrium (RA) which reduced at 3 month (RAV1: 53 ± 26 vs. 28 ± 18 mL/m\textsuperscript{2}, p < 0.001) with decreased p-wave dispersion (57 ± 20 vs. 51 ± 19 ms, p < 0.05). However, incomplete RAR was detected in 27 patients (54%) at 3 month. As a group, they were older with higher pulmonary systolic pressure, more severe tricuspid valve regurgitation and larger RA than those with normalization of RA size. Of note, the decrease of p-wave dispersion only occurred in patients with RAR. Both pre-closure RAV1 [odds ration (OR):1.057, p = 0.01] and p-wave dispersion at 3-month (OR: 1.047, p = 0.01) were the independent predictors of incomplete RAR. ROC curve revealed that p-wave dispersion ≥45ms at 3-month was 80% sensitive and 79% specific in predicting incomplete RAR (area under the curve: 0.80, p < 0.01).
Conclusion: There was overall atrial electrical and geometric reverse remodeling after ASD closure. However, RA size normalization occurred in only half of patients at mid-term follow up. Incomplete RAR after closure could be predicted by excessive pre-closure RA dilatation at baseline and p-wave dispersion at 3-month.

B005
Smad7 as a novel therapeutic agent for hypertensive cardiac disease
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Objective: Smad7 has been reported to inhibit both fibrosis and inflammation in kidney diseases, but its effect on angiotensin II (Ang II)-induced hypertensive cardiopathy remains unknown. The present study aimed to test the hypothesis that over expression of Smad7 may have therapeutic potential for Ang II-induced hypertensive cardiac remodeling.

Method: Hypertensive cardiopathy was induced in mice by subcutaneous infusion of Ang II (1.46mg/kg/day) for 28 days. Protective role of Smad7 in Ang II-mediated cardiac injury was examined by transferring Smad7 gene using an ultrasound-micro bubble-mediated system (100ug/mouse) on day 0. In addition, cardiac fibrosis and inflammation were examined by real-time PCR, western blotting and immunohistochemistry.

Result: Although equal levels of high blood pressure were developed in both Smad7-treated and control mice, Smad7 gene therapy protected against the fall in ventricular ejection fraction (EF) by 6.7% (p<0.01), which was associated with inhibition of TGF-β/Smad3-mediated cardiac fibrosis and NF-κB-driven inflammation when compared to the control-treated mice (all P<0.05, respectively).

Conclusion: Smad7 plays a protective role in Ang II-mediated cardiopathy and may be a novel therapeutic agent for hypertensive cardiovascular diseases.