S66 IS HOSPITAL READMISSION AN APPROPRIATE OUTCOME MEASURE FOR COMMUNITY-ACQUIRED PNEUMONIA?

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Introduction and Objectives There is no ideal outcome measure for studies in community-acquired pneumonia (CAP). Readmission to hospital within 30 days is one proposed measure which coincidentally the NHS now imposes financial penalties on Trusts for. We sought to investigate the frequency and validity of this measure for CAP and to examine factors predictive of readmission in CAP.
Methods All adult cases (ICD10 J10–J18) admitted in 2010 were identified from Trust Information records. Those readmitted within 30 days of discharge were regarded as cases and reason for readmission was ascertained. The two consecutive admissions after each readmission case were used as controls to identify features predictive of readmission. All were validated as CAP by inspection of radiographs and case records.
Results 562 cases were identified. 93 were excluded. 96 (20%) of the remaining 469 died. 55 (12%) were readmitted. Eight of these were excluded and six case notes were lost leaving 41 cases who were compared with 72 controls who had not been readmitted. Of these 113, mean age was 61 (95% CIs 57–65), 59% were male, 85% had one or more comorbid disease, 83% were admitted from their own home, 54% were CURB65 0–1, 26% CURB65 2, 23% CURB65 3–5. Readmission was considered by the admitting physician to be CAP-related in only 16 (39%), but even in these CRP was raised at readmission in only 81%. Non-CAP reasons for readmission were varied and were distributed across at least 8 disease areas. Only 13 (32)% of all readmissions were considered to have been preventable at the first admission. Age (OR 0.995; 95%CI 0.959 to 1.033), presence of comorbid disease (2.045; 0.415 to 10.089), Charlson comorbidity index (1.082; 0.785 to 1.491), initial length of stay (1.009; 0.984 to 1.034), CURB65>0 (2.003; 0.551 to 7.282) and initial treatment with tazocin (2.502; 0.804 to 7.784) were significantly related to readmission, but CIs all included unity.
Conclusion The low frequency, lack of relationship to CAP in the majority and lack of preventability at the index admission suggest that readmission within 30 days of discharge is not a valid outcome marker for CAP. Age and markers of biological unfitness predict readmission.

Linking mechanisms to prognosis in pulmonary arterial hypertension

S67 CHARACTERISATION OF THE ENDOTHELIAL OUTGROWTH CELL

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The existence of cells circulating in the peripheral blood with the capacity of an endothelial progenitor cell (EPC) remains controversial. This is best exemplified by the original cell posited by Asahara and colleagues to be an EPC in 1997,[1] which has now been clearly phenotyped as a monocyte with dendritic features.[2] There remains a viable candidate known as the endothelial colony-forming cell (ECFC) or endothelial outgrowth cell (OEC), named because it cannot be seen in ex vivo culture for at least 10 days. These cells proliferate in culture, form a cobblestone monocellular layer, and form networks in ex vivo assays. Given the lack of understanding of previous candidate cells we sought to better characterise the EOC. Here we present data from electron microscopy studies, immunohistochemistry, ligand stimulation studies, and mRNA microarrays that demonstrate the EOC is a true endothelial cell. Furthermore we demonstrate that these cells can potentially be used as endothelial surrogates in patients with pulmonary hypertension due to a mutation in the bone morphogenetic protein type II receptor (BMPRII). Cells taken from the peripheral blood of patients have a deficiency in intracellular signalling in the BMPRII pathway on stimulation with BMP9. This can be demonstrated by reduced phosphoSmad 1/5 protein on western blotting, and downstream with reduced Id1 gene induction by qPCR. Therefore regardless of its origin and biologically intended function, the EOC is an endothelial cell, and has the capacity to act as an easily derivable endothelial surrogate from patients in whom vascular tissue is not normally obtainable.

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S68 INHIBITION OF P38 MITOGEN ACTIVATED PROTEIN KINASE (MAPK) PREVENTS THE DEVELOPMENT OF EXPERIMENTAL PULMONARY HYPERTENSION

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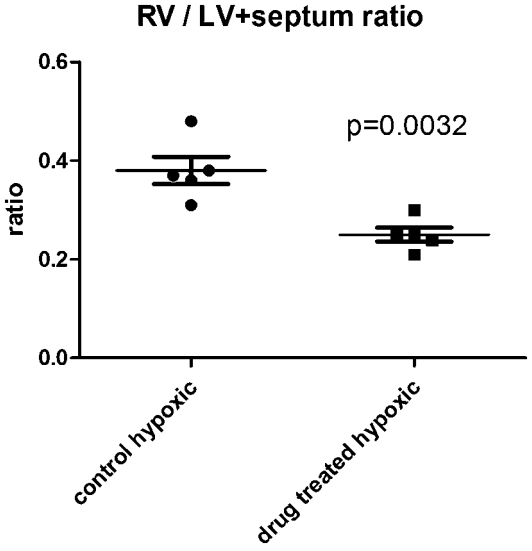
Introduction p38 MAPK has been linked to the pathobiology of pulmonary hypertension (PH) but its role has not been fully investigated. We have previously highlighted the importance of the p38 MAPK pathway in the proliferative response of pulmonary artery fibroblasts to hypoxia. In addition, the BMPR2 mutations seen in PH can lead to increased signalling through the p38 MAPK pathway. These findings suggest p38 MAPK may be an appropriate target for the treatment of PH.
Methods We undertook an in vivo prevention study on male Sprague-Dawley rats that were exposed to hypobaric hypoxia (at 550 mm Hg=10% FiO2) for a period of 2 weeks. Five animals were dosed daily via an intraperitoneal route, with SB-203580, a p38 MAPK specific inhibitor, and a further five animals acted as controls. After 2 weeks the animals had right ventricular systolic pressure (RVSP) measured,



Abstract S68 Figure 1 RV/LV+septum ratio.