Spoken sessions

Methods Secondary analysis of the Edinburgh pneumonia database, a prospective observational study of CAP (2005–2010). All discharged patients were included. Follow-up data were obtained from a database linked to national morbidity and mortality registers. Outcomes were assessed using cox proportional hazards regression adjusting for confounding variables (age, gender, previous cardiovascular events, ACE-inhibitor/anti-platelet use, smoking status and severity of pneumonia).

Results Data from 1631 patients with complete follow-up were analysed. Readmissions occurred in 728 patients (44.6%) with 157 readmissions within 30 days of discharge. 133 patients had a further hospitalisation with CAP. The 1 year mortality rate was 12.8%.

523 patients were current statin users. There were significant differences between statin and non-statin users. Statin users were older, suffering more cardiovascular disease, stroke, diabetes, renal disease, COPD and a greater severity of pneumonia.

1 year mortality rates were similar in statin and non-statin users. After adjusting for baseline differences, statins were associated with a non-significant trend towards lower 1 year mortality HR 0.78(0.55–1.1). When adjusted for the propensity score, the difference in mortality became statistically significant HR 0.70 (0.50–0.98).

In the fully adjusted analysis, statins were not significantly associated with readmissions HR 0.85(0.70–1.03) but were associated with a significantly lower risk of recurrent pneumonia HR 0.60 (0.37–0.96). There was no association with reduced cardiovascular hospitalisations HR 0.94(0.65–1.34). There were no beneficial effects seen with either antiplatelet or ACE-inhibitor use following CAP.

Conclusion Statins are associated with reduced 1 year mortality and a significantly lower rate of recurrent pneumonia. This is the first study to show that statins may improve long term outcomes in CAP, and that the associated morbidity and mortality can be modified.

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INCREASED RISK OF UPPER RESPIRATORY INFECTION WITH ADDITION OF INTERMITTENT BOLUS-DOSE VITAMIN D SUPPLEMENTATION TO A DAILY LOW-DOSE REGIMEN

¹AR Martineau, ¹Y Hanifa, ¹RL Hooper, ¹KD Witt, ¹M Patel, ¹A Syed, ¹DA Jolliffe, ¹PM Timms, ¹Z Balayah, ¹N Stevens, ²DA Clark, ¹S Eldridge, ²N Barnes, ¹CJ Griffiths; ¹Queen Mary University of London, London, United Kingdom; ²Barts Health NHS Trust, London, United Kingdom

10.1136/thoraxjnl-2013-204457.130

Introduction and Objectives Meta-analysis of clinical trials of vitamin D supplementation for the prevention of acute respiratory infection (ARI) shows a protective effect in the general population, but there is controversy regarding the optimal dosing regimen. Low-dose vitamin D supplementation is already recommended in older adults for prevention of fractures and falls, but clinical trials investigating whether higher doses could provide additional protection against ARI are lacking.

Methods We conducted a double-blind cluster-randomised placebo-controlled trial of high- vs. low-dose vitamin D supplementation in residents and staff of sheltered accommodation schemes in London, UK. 108 schemes were allocated to receive the intervention (vitamin D $_3$ 2.4 mg 2-monthly + 10 μg daily for residents; 3 mg 2-monthly for staff) or control (vitamin D $_3$ 10 μg daily for residents, nil for staff) over the course of one year. The primary endpoint of the trial was time from first dose of study

medication to date of first ARI, determined by a validated acute respiratory symptom score recorded prospectively in a symptom diary. Secondary outcomes included time to first upper / lower respiratory infections (URI/LRI) and mean serum 25-hydroxyvitamin D (25[OH]D) concentration.

Results 240 participants were included in the intention-to-treat analysis (137 participants in 54 schemes allocated to intervention, mean baseline 25[OH]D 43.8 nmol/L vs. 103 participants in 54 schemes allocated to control, mean baseline 25[OH]D 43.8 nmol/L). Median time to ARI was 203 days in the intervention arm and 227 days in the control arm (adjusted HR 1.18, 95% CI 0.80 to 1.74, p = 0.42). Allocation to the intervention arm of the trial was associated with increased risk of URI (adjusted HR 1.48, 95% CI 1.02 to 2.16, p = 0.04), but not with altered risk of LRI (adjusted HR 1.12, 95% CI 0.73 to 1.70, p = 0.61). Mean serum 25(OH)D concentration at 1 year was 84.8 nmol/L vs. 58.5 nmol/L in intervention vs. control arms (p < 0.0001).

Conclusions Addition of intermittent bolus-dose vitamin D supplementation to a daily low-dose regimen improved vitamin D status in older adults and their carers, but it did not influence risk of ARI, and was less effective at preventing URI.

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DERIVATION AND VALIDATION OF THE BRONCHIECTASIS SEVERITY INDEX: AN INTERNATIONAL MULTICENTRE OBSERVATIONAL STUDY

¹JD Chalmers, ²P Goeminne, ¹S Aliberti, ³M McDonnell, ⁴S Lonzi, ³J Davidson, ¹L Poppelwell, ¹W Salih, ⁴A Pesci, ²L Dupont, ¹TC Fardon, ³A De Soyza, ⁵AT Hill; ¹University of Dundee, Dundee, UK; ²University Hospital Gasthuisberg, Leuven, Belgium; ³University of Newcastle, Newcastle, UK; ⁴University of Milan Bicocca, Monza, Italy; ⁵Royal Infirmary of Edinburgh, Edinburgh, UK

10.1136/thoraxjnl-2013-204457.131

Introduction There are no risk stratification tools for morbidity and mortality in bronchiectasis. As more treatments become available, it is important to identify patients at risk of exacerbations, hospital admissions and mortality to target novel therapies.

Methods A prospective observational study at a specialist bronchiectasis clinic in Edinburgh, UK was used to derive a bronchiectasis severity index using cox-proportional hazards regression to identify independent predictors of mortality and hospital admission over 4 years follow-up. Averaged \mathcal{B} -coefficients were used to award points for each independent variable and the discrimination of a derived score was tested using the area under the receiver operator characteristic curve (AUC). The score was validated in independent cohorts from Dundee, UK (N = 218), Leuven, Belgium (N = 253), Monza, Italy (N = 105) and Newcastle, UK (N = 126).

Results 608 patients were included in the derivation cohort. Independent predictors of future hospital admissions were prior hospital admissions hazard ratio (HR) 13.5 (9.40–19.46), MRC dyspnoea score > 4, HR 2.42 (1.66–3.52), FEV $_1$ <30% predicted HR 1.52 (1.03–2.25), Pseudomonas aeruginosa colonisation HR 2.16 (1.36–3.43), colonisation with other organisms HR 1.66 (1.12–2.44) and > 3 lobes involved on HRCT HR 1.48 (1.02–2.15). In the model for mortality, independent predictors were Age >70 years 8.57 (1.15–63.63), FEV $_1$ <30% predicted HR 4.47 (1.60–12.53), prior hospital admissions HR 2.43 (1.30–4.53) and 3 or more exacerbations per year prior to the study HR 2.03 (1.02–4.03).

A64 Thorax 2013;68(Suppl 3):A1–A220