suggesting an altered inflammatory phenotype, at least in these models, to these stimuli.

REFERENCES

Abstract P159 Figure 1.

**P160** RETROSPECTIVE STUDY ANALYSING WHETHER OPTIMIZATION OF PULMONARY VASCULAR ENHANCEMENT INFLUENCES DIAGNOSTIC OUTCOME IN THE INTERPRETATION OF CT PULMONARY ANGIOGRAMS (CTPA)

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Introduction and Objectives Pulmonary embolism (PE) is a common cardiovascular emergency and is the most common preventable cause of hospital deaths. Various factors can impact on the diagnostic accuracy of CTPA, which is now considered the 1st line investigation for PE diagnosis. Mis-timing of contrast medium administration or cardiac impairment can result in an indeterminate scan through hidden or mimicked emboli. This study focuses on the accuracy of CTPAs in excluding a diagnosis of PE.

Method This is a single centre study looking at all inpatient and outpatient CTPAs carried out over a 12-month period with matched CTPA and lower limb ultrasound Doppler events selected. Of these matched studies, all positive CTPAs for PE were excluded. Of the remainder, only those studies done within 6 months of the original CTPA were included in the study. CT pulmonary arterial (PA) opacification with intravenous contrast medium administration or cardiac impairment can result in an indeterminate scan through hidden or mimicked emboli. This study focuses on the accuracy of CTPAs in excluding a diagnosis of PE.

Method This is a single centre study looking at all inpatient and outpatient CTPAs carried out over a 12-month period with matched CTPA and lower limb ultrasound Doppler events selected. Of these matched studies, all positive CTPAs for PE were excluded. Of the remainder, only those studies done within 6 months of the original CTPA were included in the study. CT pulmonary arterial (PA) opacification with intravenous contrast medium administration or cardiac impairment can result in an indeterminate scan through hidden or mimicked emboli. This study focuses on the accuracy of CTPAs in excluding a diagnosis of PE.

Results From the 32 CTPAs included in the study, 28 had a negative initial CTPA with subsequent negative CT or US follow-up. 4 of these cases had an initial negative CTPA with a subsequent positive CTPA or lower limb US Doppler study. Of these latter cases only one initial CTPA was deemed poorly suboptimal (average 174HU) with optimal pulmonary arterial opacification in all other three initial CTPAs.

Conclusion This study demonstrates that despite a large number of initially negative CTPAs undergoing subsequent follow-up imaging due to presumed persistent or recurring patient symptoms, the majority (87.5%) of these negative scans were still negative and only 12.5% were positive for PE. No major pitfall has been identified in local CTPA acquisition technique which may have led to subsequent misses of potentially life-threatening PEs.

**P161** A RANDOMISED CONTROLLED TRIAL OF SINGLE COMBINATION Budesonide/Formoterol INHALER AS MAINTENANCE AND RELIEVER THERAPY IN ASTHMA PATIENTS AT RISK OF SEVERE EXACERBATIONS

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Introduction The Single budesonide/formoterol inhaler Maintenance And Reliever Therapy (SMART) regimen reduces severe asthma exacerbations, but it is uncertain whether it increases the risk of adverse effects due to high beta-agonist and corticosteroid doses with both short-term and cumulative exposure.

Objectives Our hypothesis was that treatment with the SMART regimen would reduce the risk of beta-agonist overuse but that when such episodes occurred, patients were less likely to seek medical review and, that any reduction in severe exacerbations would be at the cost of a higher systemic corticosteroid burden.

Methods This was a 24-week, open-label, parallel group trial in 303 asthma patients with a recent exacerbation, conducted at four primary healthcare practices and one secondary care hospital. Participants were randomised to 200/6μg budesonide/formoterol metered dose inhaler according to the SMART regimen (two actuations twice daily maintenance with one extra actuation as-needed) or a fixed-dose regimen (two actuations twice daily maintenance) with one to two actuations of 100μg salbutamol as-needed (‘Standard’), with electronic monitoring of actual inhaler use. The primary outcome was the proportion of participants with at least one high beta-agonist use episode (>12 budesonide/formoterol actuations/day in SMART or >16 salbutamol actuations/day in Standard).

Results There was no significant difference between groups in the proportion of participants with at least one high use episode: SMART 84/151 (56%) vs Standard 68/152 (45%); relative risk (95% CI) 1.24 (0.99–1.56), p = 0.058. There were fewer days of high use [mean (SD) 5.1 days (14.3) vs 8.9 days (20.9)], relative rate (RR) (95% CI) 0.58 (0.39–0.88), p = 0.04] and days of high use without medical review [8.5 days (17.8) vs 18.3 days (24.8)] per high use patient, RR 0.49 (0.31–0.75), p = 0.001 in the SMART group. The SMART regimen resulted in higher average daily inhaled corticosteroid exposure, but reduced oral corticosteroid exposure, with no difference in composite systemic corticosteroid exposure [ratio of means (95% CI) 1.03 (0.86–1.22), p = 0.76]. SMART participants had fewer severe asthma exacerbations [35 vs 66, RR 0.54 (0.36–0.82), p = 0.004].

Conclusions The SMART regimen has a favourable risk/benefit profile in patients at risk of severe asthma exacerbations.

**P162** EFFECT OF ADDING PROPRANOLOL OR INCREASED INHALED CORTICOSTEROID DOSE IN PERSISTENT ASTHMA
Introduction and Objectives Chronic propranolol does not improve airway hyper-responsiveness (AHR) in persistent asthma taking medium dose inhaled corticosteroid (ICS), 440μg/day. We wished to assess for any putative corticosteroid-sparing effect of propranolol added to low dose ICS versus higher dose ICS, on histamine AHR.

Methods We conducted a randomised double-blind placebo-controlled crossover trial in mild-moderate persistent asthmatics. Patients were run-in for 2 weeks on hydrofluoroalkane-beclomethasone dipropionate (HFA-BDP) 100μg/day. Patients were then randomised to either: propranolol 80mg/day plus HFA-BDP 100μg/day; or placebo plus HFA-BDP 400μg/day; each for 4 weeks. Propranolol was up-titrated to 80mg/day for the second 2 weeks of treatment. Patients received tiotropium 18μg/day during run-in and both treatments, which was subsequently discontinued 5 days prior to histamine challenge (primary outcome).

Results 16 patients completed, mean: age 38yr; FEV1 86.4%; Histamine PC20 1.39mg/ml; ICS 406μg/day. Histamine PC20 remained unchanged adding propranolol to HFA-BDP100 compared to baseline (HFA-BDP100): 0.17 doubling dilution (dd) difference (95%CI -0.58–0.92), but there was a significant improvement with HFA-BDP400 compared to both baseline 1.05dd (95%CI 0.43–1.66), P = 0.02; and propranolol 0.88dd (95%CI 0.45–1.30), P = 0.006 (Figure 1a). Significant improvements from baseline were observed with HFA-BDP400 for exhaled nitric oxide, blood eosinophils (Figure 1b) and Asthma Quality of Life Questionnaire (AQLQ) symptom score (Figure 1c), but not with propranolol. Salbutamol recovery time post-challenge was partially blunted by propranolol (median prolongation 3min compared to both HFA-BDP400, P = 0.002). Domiciliary evening FEV1 also fell with propranolol (mean reduction from baseline 0.22L [95%CI 0.10–0.34L], P = 0.012) while Asthma Control Questionnaire (ACQ) showed no significant changes with either treatment compared to baseline.

Conclusions In mild-moderate persistent asthmatics, propranolol produced no additive effects on top of low dose ICS, while further significant improvements in AHR and inflammation were seen with a higher dose of ICS. Propranolol attenuated salbutamol recovery and reduced evening FEV1, but not ACQ or AQLQ.

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P162 Effect of adding propranolol or increased inhaled corticosteroid dose in persistent asthma

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