JOURNAL CLUB SUMMARIES

What's hot that the other lot got

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CAN HIGH FLOW NASAL CANNULA PREVENT INTUBATION?

A systematic review and meta-analysis compared conventional care and non-invasive ventilation (NIPPV) with the use high-low nasal cannula (HFNC) in adults with respiratory failure (Monro-Somerville *et al*. *Crit Care Med* 2017;45:e449–56). Fourteen trials were included in the systematic review, nine in the meta-analysis (2507 subjects). There was no difference in mortality (HFNC, 60/1006 (6%) vs usual care, 90/1106 (8.1%)) (n=2112; p=0.29; OR, 0.83; 95%CI, 0.58 to 1.17). There was no difference in rate of intubation rate (HFNC, 119/1207 (9.9%) vs usual care, 204/1300 (15.7%)) (n=2507; p=0.08; OR, 0.63; 95%CI, 0.37 to 1.06). There was improved patient comfort and dyspnoea.

WHAT ARE THE LONG TERM IMPLICATIONS OF UNDIAGNOSED COPD?

Screening for COPD occurs in previous or current smokers who present with respiratory symptoms. A group from Denmark carried out a prospective cohort study from the Copenhagen General Population Study, to see if there was an increased rate of exacerbations, pneumonia and death, those with undiagnosed COPD (Çolak *et al*. *Lancet Respir Med* 2017;5:426–34). A total of 95 288 people between November 2003 and July 2013 were screened. A high risk group (aged over 40, at least 10 pack years of tobacco, no previous history of asthma) of 32 518 (34%) were identified. Of these 2903 (78%) had undiagnosed COPD and 2052 (71%) were symptomatic. Over a median period of follow-up of 6.1 years, in the high risk population there were 800 exacerbations of COPD, 2038 episodes of pneumonia, 2789 deaths, 152 due to respiratory disease. When compared with those not at risk of COPD the adjusted HR was 5.0 (95% CI 2.8 to 8.9) for exacerbations, 1.7 (95% CI 1.3 to 2.2) for pneumonia, 0.7 (95% CI 0.2 to 3.0) for death from respiratory causes, and 1.3 (95% CI 1.1 to 1.6) for death. For those who were symptomatic the HRs were higher being 15.5 (95% CI 11.0 to 21.8) for exacerbations, 2.8 (95% CI 2.4 to 3.3) for pneumonia, 4.3 (95% CI 2.8 to 6.7) for death from respiratory causes, and 2.0 (95% CI 1.8 to 2.3) for death from all causes in COPD.

POTENTIAL THERAPIES FOR CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION (CTEPH)

This paper assessed the benefit and safety of balloon angioplasty for CTEPH (Ogo *et al*. *Eur J Radiol* 2017;89:270–6). A series of 80 consecutive patients with inoperable CTEPH underwent Balloon Pulmonary Angioplasty (BPA) guided by cone-beam CT or ECG gated area detector CT (ADCT). A total of 385 sessions of BPA was carried out for lesions within the sub-segmental pulmonary arteries (1155), segmental pulmonary arteries (738 lesions) and lobar arteries (4). There were significant improvements seen in 6 min walk, brain natriuretic peptide level, exercise capacity and pulmonary haemodynamics observed at 3 months and 1 year. There were no cases of death or cardiogenic shock. The rate of wire perforation was 0.3% and reperfusion pulmonary oedema was 0.3%. This suggests the procedure is safe and shows good symptomatic improvement in a cohort of patients with inoperable disease.

CAN THE RATE OF AUTOPLEURADESIS WITH AN INDWELLING PLEURAL CATHETER BE IMPROVED?

Patients with indwelling pleural catheters (IPC) for malignant pleural effusions suffer significant morbidity, mortality and social disruption from having these. The ideal is the free from the IPC achieving autopleuradesis. A group of 149 patients with an IPC in place for a malignant pleural effusion were randomised to daily drainage (73) or standard drainage every other day (76) (Wahidi *et al*. *Am J Respir Crit Care Med* 2017;195:1050–7). The outcome being a complete or partial response based on radiographic and symptomatic changes. The rate of autopleuradesis was greater in the daily drainage arm compared with the conventional arm (47% vs 24% p=0.003), the median time was shorter (54 day; 95% CI, 34 to 83) as compared with the standard arm (90 day; 95% CI, 70 to non-estimable). There was no difference in adverse events quality of life or patient satisfaction.

IS THERE GENETIC HETEROGENEITY WITHIN LUNG TUMOURS?

The greater the genetic diversity of a tumour the greater the potential for natural selection and tumour evolution (Jamal-Hanjani *et al*. *N Engl J Med* 2017;376:2109–21). Tumour samples of 100 chemotherapy naive patients with non-small cell lung cancer stages IA to IIIA were collected. Whole genome sequencing of the tumour samples was carried out. Within the tumour there was median of 30% (range, 0.5 to 93) of somatic mutations identified as sub clonal and a median of 48% (range, 0.3 to 88) of copy-number alterations. The rate of clonal mutations was greater in squamous cell carcinoma than adenocarcinoma (p=0.003), this is potentially related to smoking, median 32 pack years adenocarcinoma, 41 pack years squamous cell. In adenocarcinoma the rate of clonal and sub clonal mutation was higher in smokers and the higher the proportion of sub clonal mutations the higher the risk of death (>48%, cohort median). Further intratumour heterogeneity was assessed by looking for mutation clusters. There were 525 identified with a median of 5 per tumour, most (86%) were sub clones from the same genetic tree. The accumulation of early mutations was shown to be due to tobacco smoking and quantity. In those who had not smoked for a number of years (>20) a late clonal signal was identified suggesting tumour latency of many years. The driver mutations for the tumours were shown to be in EGFR, MET, BRAF, and TP53. Later heterogeneous driver mutations were found in 75% of the tumours in genes that are involved in chromatin modification and DNA damage and repair. Overall the greater the heterogencity of a tumour the greater the risk of death or recurrence (HR, 4.9; 95% (CI, 1.8 to 13.1; $p=4.4\times10^{-4}$) the median to time of death or recurrence in the high risk group being 24 months.

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