

**JOURNAL CLUB SUMMARIES** 

# What's hot that the other lot got

Flizabeth Batalla-Duran

## TROPONIN PREDICTS PROGNOSIS IN COMMUNITY-ACOUIRED PNEUMONIA

It is not unusual for high sensitivity cardiac troponin (cTnT) to be measured when a patient presents to hospital with chest symptoms. Often the result can be slightly raised despite the primary problem not being cardiac. It seems logical that a severe pneumonia may cause a troponin leak by causing strain on the heart. This study by Vestiens et al (Respirology. http://dx.doi. org/10.1111/resp.12996) sought to see if elevated troponin is a prognostic marker in patients admitted with community-acquired pneumonia (CAP). The study looked at cTnT levels on admission in 295 patients admitted with CAP. Levels were elevated (>14 ng/L) in 45%. Short-term (30 days) and long-term (up to 4.1 years) mortality were significantly higher in those with elevated cTnT. Elevated cTNT >28 ng/L was found to be a strong independent risk factor for mortality both short-term (OR 21.9, 95% CI 4.7 to 101.4) and long-term (OR 10.7, 95% CI 5.0 to 22.8). The study also combined troponin levels with the Pneumonia Severity Index (PSI) and found this was a stronger predictor of short-term mortality than PSI score alone.

### STATINS IN THE TREATMENT OF LUNG CANCER

Statins have received bad press in recent years. However, experimental and observational studies suggest they may have additive effects with chemotherapy to impair tumour growth, delay relapse and prolong life in some cancer types. This large UK double-blinded randomised placebo-controlled trial (J Clin Oncol. http://dx.doi.org/10.1200/JCO.2016.69. 7391) looked to investigate any potential benefits of adding daily pravastatin 40 mg to standard care chemotherapy (etoposide plus cisplatin or carboplatin) in 846 patients with small cell lung cancer. The primary end point was overall survival (OS) with secondary end points of progressionfree survival (PFS), response rate and toxicity. Unfortunately, although well tolerated,

Correspondence to Dr Elizabeth Batalla-Duran, ST6, Royal Devon and Exeter Hospital, Barrack Road, Exeter EX2 5DW, UK; ebatalla-duran@nhs.net there was no benefit found of adding pravastatin, in either limited or extensive disease. The 2-year OS was 13.2% for added pravastatin vs 14.1% for placebo (HR 1.01). Median OS was 10.6 vs 10.7 months, while median PFS was 7.7 months (pravastatin) vs. 7.3 months (placebo).

# WHY DO PEOPLE USE E-CIGARETTES? ASK TWITTER

There has been an exponential rise in the use of electronic nicotine delivery systems (ENDS) in the past 5 years. Are all users trying to quit tobacco products (combustibles) or do they have other reasons for 'vaping'? This study in PLoS ONE (http:// dx.doi.org/10.1371/journal.pone.0170702) looked to social media to find out. They analysed all tweets on Twitter between 2012 and 2015 that had references to using e-cigarettes (using search terms such as vaping, e-cigarette and their derivatives) to see if there were clear reasons for people taking up ENDS. Adverts and tweets not referencing actual use were excluded. Using a target sample of 2900 tweets per year, the authors found that initially, in 2012, by far the most common reason for ENDS use appeared to be quitting combustibles (43%), followed by social image (21%) and use indoors (14%). Safety compared with combustibles was cited in 9%. Other reasons cited were flavour, favourable odour and cost. Interestingly, by 2015 there had been a considerable shift in the reasons cited. Somewhat concerning, social image became the most cited reason (37%), while quitting combustibles and use indoors declined to 29% and 12%, respectively. There have been more restrictions on use of e-cigarettes indoors as concerns about their side effects grow and this may explain some of the decline in the latter. The authors discuss that using social media to monitor behaviours can be used in public health surveillance and to guide future health plans.

# THE EFFECT OF THROMBUS LOCATION ON MORTALITY

Central pulmonary emboli with haemodynamic compromise are recognised to have high mortality and the NICE guidance is to consider thrombolysis in these patients. This study (*Cardiol J.* http://dx.

doi.org/10.5603/CJ.a2017.0021) looked to determine if thrombus location in haemodynamically stable patients had an effect on 30-day mortality. It also looked at whether choice of treatment of central thrombus (unfractionated heparin (UFH), low molecular weight heparin (LMWH) or thrombolysis) affected outcome. Eight hundred and seventy-four normotensive patients with acute PE were included and grouped according to PE location central (saddle or main pulmonary artery) (319), lobar (264) or distal (291). Seventy-four patients died in the 30-day follow-up period. Analysis showed right ventricular dysfunction on echocardiography and a raised troponin were independent predictors of mortality (p<0.001). Although mortality was higher in patients with central thrombus, multivariate analysis did not find it to be an independent predictor of mortality. There was no difference in mortality rate between treatment with LMWH and UFH. Traditionally, UFH may be used in stable patients with large PE in case they deteriorate and require thrombolysis. However, this study suggests that the simpler to use LMWH is just as acceptable.

#### **COCHRANE NEWSFLASH**

In cystic fibrosis, drugs that may correct the underlying molecular defect caused by mutation of the cystic fibrosis transmembrane conductance regulator (CFTR) gene are of great interest and one such agent, ataluren, has the potential to overcome mutations that cause premature truncation of the CFTR protein. A Cochrane Review, including one phase III trial (238 patients) did not demonstrate significant improvement in key outcomes, including respiratory function, and showed ataluren was associated with a significant risk of renal impairment (relative risk 17.7 (95% CI 2.4 to 130.5)). A post hoc analysis excluding patients on nebulised tobramycin did show favourable results for respiratory function outcomes, and this has resulted in an ongoing clinical trial.

**Competing interests** None declared.

**Provenance and peer review** Commissioned; internally peer reviewed.



To cite Batalla-Duran E. Thorax 2017;72:488.

*Thorax* 2017;**72**:488. doi:10.1136/thoraxjnl-2017-210255

#### **REFERENCES**

1 Aslam AA, Higgins C, Sinha IP, et al. Ataluren and similar compounds (specific therapies for premature termination codon class I mutations) for cystic fibrosis. Cochrane Database Syst Rev 2017;(1):CD012040.