Is investigation of patients with haemoptysis and normal chest radiograph justified?

We read with interest the article by Thirumannar et al. They described their experience of 270 consecutive patients referred with haemoptysis and a normal chest radiograph. In their study they found the incidence of respiratory malignancy within this group was 9.6% (26 individuals) and of these 22 were primary lung malignancies. This is slightly higher than the 3–6% incidence previously reported in the literature.2–4

We have delivered a nurse-led clinic for patients referred via the 2 week wait system with haemoptysis and a normal chest radiograph. Patients referred for mostly by infection or an idiopathic cause. Approximately 10% of people referred for investigation were awaiting a change in clinical presentation prior to admission to the clinic. Variables within the haemoptysis clinic. Variables within the non-localising chest x-rays included increased cardiothoracic ratio, bullae and hyperinflated lung fields. One chest x-ray report suggested that the ‘bulky’ mediastinal appearance was likely to be due to rotation. Persistent haemoptysis (duration of >1 week) was seen in 15 of the 25 lung cancer patients but was also seen in 145 of the 325 patients with a benign diagnosis. Duration of haemoptysis is therefore not a good predictive factor of risk for malignancy and this further supports the work of the previous authors.

Haemoptysis is a fairly common, usually self-limiting symptom which is accounted for mostly by infection or an idiopathic cause. Approximately 10% of people referred with this symptom will not actually have haemoptysis. We reviewed our lung cancer patients retrospectively, and found that a previous history of any malignancy was important and should be added to the risk stratification algorithm. The clinic ceased to run in January 2009 and, to date, no patients have been re-referred or appeared on the cancer database. This would also suggest that our model is a safe, efficient method of screening patients with a possible thoracic malignancy and freeing up resources in the urgent 2 week wait clinic.

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REFERENCES


The learning curve for EBUS-TBNA

We read with interest the paper by Kemp and colleagues which utilises cumulative sum (CUSUM) to analyse the learning curves associated with endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA). The retrospective study from five centres demonstrated that variable learning periods are required to attain proficiency in the procedure, and a pooled sensitivity of 67.4% was observed.

The authors are to be commended on using CUSUM to calculate the learning curves for EBUS-TBNA; however, several points deserve comment. First, the study only includes patients undergoing EBUS-TBNA for the diagnosis or staging of lung cancer. In clinical practice, the procedure is also commonly employed for the diagnosis of isolated mediastinal lymphadenopathy, and these procedures should be incorporated in the learning process. Secondly, the authors included non-malignant nodes in the CUSUM analysis. Therefore, it may be possible to inadequately sample a benign node and for the result to be assigned as a true negative. This highlights the importance of reporting the disease prevalence for each cohort. Thirdly, utilising the criteria employed in this paper, there is potential to

### Table 1: common diagnostic causes of haemoptysis

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer</td>
<td>23</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>52</td>
</tr>
<tr>
<td>Infection</td>
<td>107</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>70</td>
</tr>
<tr>
<td>ENT</td>
<td>24</td>
</tr>
<tr>
<td>Cardiac</td>
<td>6</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>5</td>
</tr>
<tr>
<td>Not haemoptysis</td>
<td>34</td>
</tr>
<tr>
<td>Anticoagulation therapy</td>
<td>8</td>
</tr>
<tr>
<td>Vascularis</td>
<td>4</td>
</tr>
<tr>
<td>DNA follow-up</td>
<td>13</td>
</tr>
<tr>
<td>Died</td>
<td>2</td>
</tr>
</tbody>
</table>

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Smokers commonly misperceive that nicotine is a major carcinogen: National survey data

In vitro testing has shown that nicotine may play a role in making cancers more aggressive, but the currently available evidence does not suggest that nicotine in itself induces cancer. Despite this, many smokers believe that nicotine causes cancer. For example, in a USA-based study it was found that 65% of smokers believed nicotine causes lung cancer and 71% believed it caused oral cancer. Furthermore, some smokers regard nicotine replacement therapy (NRT) as also being carcinogenic. These findings are concerning since misperceptions about nicotine may result in underutilisation of NRT. Therefore, we aimed to assess these views in New Zealand (NZ) smokers, with the context being a country in which NRT is provided in a heavily subsidised form and widely distributed via the national quitline service.

Data were collected through the NZ arm of the International Tobacco Control Policy Evaluation Survey (ITC Project) which derives its sample of smokers from the NZ Health Survey (a representative national sample). From this sample we surveyed adult smokers in two survey waves (n=1576 and n=923) 1 year apart (with wave 2 in 2008/early 2009). Here we focus on those who completed both surveys (to facilitate comparisons over time). Further details of the methods, including response rates, attrition and weighting processes, are available in online reports (at: http://www.nihr.ac.nz/tcproject.html).

When asked if ‘the nicotine in cigarettes is the chemical that causes most of the cancer?’, most smokers in wave 1 (52.6%) said that it was true, 36.7% said it was false (the correct answer) and 10.7% could not say. The proportion answering ‘true’ was fairly similar in wave 2 at 52.1%. In a multivariate model (that adjusted for demographics, socioeconomic position, mental health and smoking-related beliefs and behaviours), certain groups of smokers were significantly more likely to believe that nicotine was carcinogenic. These included older smokers (≥50 vs <50 years); Māori smokers (vs European/other, adjusted OR = 1.77, 95% CI 1.22 to 2.58); and Asian smokers (vs European/other, aOR=3.25, 95% CI 1.35 to 7.53). One of two forms of financial stress was significantly associated with this misperception (aOR=1.57, 95% CI 1.03 to 2.41 for not spending on household essentials) but the individual and small area deprivation measures were not. Of 13 other variables considered (covering mental health, smoking beliefs and behaviours), only having a higher AUDIT score (reflecting an increased risk of hazardous alcohol use), was significantly associated with this misperception.

The finding that smokers in this national sample commonly have misconceptions about the carcinogenicity of nicotine is consistent with findings from the USA and the UK. This population of smokers also commonly have misperceptions around the relative harmfulness of ‘lights’, ‘roll-your-own’ tobacco, menthols and smokeless tobacco. How best to address all such misperceptions is complex, but at least for the nicotine and cancer issue evaluation work could be considered on: (1) inclusion of this information as part of warning labels on tobacco packets; and/or (2) mass media campaigns that highlight the relatively safety (and effectiveness) of NRT.

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