Endobronchial tumours in a campaign for early detection of bronchial cancer: Computed tomography versus endoscopy

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KEYWORDS
Lung cancer; Screening; CT; Bronchoscopy

Abstract
Objective: To study endobronchial cancers occurring in a population at high risk of bronchial cancer (history of surgically treated bronchial or ENT cancer in complete remission, and symptoms due to smoking) detected by annual volume CT scans and biannual fibroscopy.
Material and methods: Two hundred and sixty-six patients were included in this single centre prospective study; 27 bronchopulmonary cancers were detected. Ten endobronchial cancers (37%) were identified by fibroscopy (nine invasive cancers and one carcinoma in situ) in 10 patients (nine men) (51–78 years old) nine of whom were smokers (dark tobacco: seven). The screening CTs were reappraised by two radiologists.
Results: Three cancers out of 10 were detected by CT during the initial reading. The sensitivity of the reappraised CT was 80% with seven false positives. In five cases, the mean period between the first CT scan where the lesion was visible retrospectively, but not described, and the diagnostic fibroscopy was 463 days (213–808 days); two cancers were not visible in the CT scan. Seven curative treatments were undertaken.
Lung cancer is the prime cause of death by cancer in the world [1]. Several studies have shown that helical computed tomography with a low radiation dose is a more sensitive technique than chest X-ray for detecting small bronchial cancers [2—11]. The cancers detected in these studies were largely intraparenchymatous, with peripheral adenocarcinoma clearly predominating, being detected at a rate between 0.4 and 2.7% [2—11]. This early detection provides hope for reducing mortality specifically due to bronchial cancer owing to management starting at a stage which can be cured by surgery [12]. The recently published NLCT randomised study shows a reduction in the specific mortality due to lung cancer of 20.3% in the group screened by CT scan compared with the X-ray screened group, and a reduction of 6.9% in all-cause mortality in the CT group [13]. These results raise great hopes but must be confirmed [13]. Despite the most rigorous reading possible of the examinations, certain cancers are not detected in a CT scan. The proportion of cancers ignored in CT has been little reported in the literature. White et al. reported 14 cases of undiagnosed cancers in 37,500 chest CT scans: an endobronchial location was found in each case [14]. Endobronchial cancers, less common than peripheral cancers, are usually discovered by fibroscopy, performed exceptionally in screening programmes for high-risk patients [15]. White light bronchoscopy is the usual technique for detecting cancers with a central location. Autofluorescence bronchoscopy allows preneoplastic lesions and carcinomas in situ to be detected with greater sensitivity than white light bronchoscopy [16,17].

The role of CT scans in detecting endobronchial cancers has been little studied to date [18].

Our prospective study has evaluated the usefulness of combining CT with white light and autofluorescence fibroscopy for detecting bronchial cancers early, in the context of an annual screening programme for bronchopulmonary cancer in patients with a very high risk of the disease. The aim of our study was to analyse the CT scans of endobronchial lesions identified by fibroscopy, the standard technique for identifying proximal endobronchial tumours.

**Material and methods**

**Patients**

Two hundred and sixty-six patients were included in the Biomarkscan protocol between 2001 and 2009. This study had been approved by the university hospital’s ethics committee (DGS 2002/0247) and all the participants had given their informed consent in writing.

The inclusion criteria (Table 1) were as follows:

- patients with a history of operated non-small-cell lung cancer in complete remission;
- patients with a history of ENT cancer in remission;
- patients with preneoplastic bronchial lesions;
- patients who were active smokers or who had stopped smoking (more than 20 pack-year) with respiratory symptoms (cough or dyspnoea) but no clinical signs of lung cancer.

The exclusion criteria (Table 1) were as follows:

- unstable cardiac insufficiency or respiratory insufficiency;
- patients who could not be monitored annually;
- anticoagulant treatment or pathology with a high risk of haemorrhage;
- history of cancer within the last 5 years (except for non-melanomatous skin cancers, non-small-cell cancers of the lungs and ENT cancers in remission);
- a history of thoracic radiotherapy.

The patients included in the Biomarkscan protocol agreed to be monitored for 5 years. All the patients had:

- a reference low-dose chest CT scan which was repeated annually;
- white light and autofluorescence fibroscopy in years 1, 3 and 5;
- cytological analysis of induced sputum in years 2 and 4;
- annual blood analysis to search later for biomarkers.

On inclusion, a questionnaire including the personal details of the patient, his or her medical history, the quantity and type of tobacco smoked, exposure to other physical or chemical risk factors that could induce bronchial cancer, and tracheobronchial functional signs, was completed by the doctor and patient. In the 266 patients included, 27 bronchopulmonary cancers were identified, i.e. a prevalence of 10.15%. From these, we isolated a sub-group of 10 patients (nine men and one woman), with a mean age of 62.2 years (51—78), in whom fibroscopy showed a cancerous lesion with an endobronchial location.

**CT technique**

The CT examinations were performed with a 16-slice CT scanner (Siemens Sensation 16; Siemens AG, Medical Solutions, Erlangen, Germany). The acquisition parameters were as follows: collimation of 0.75 mm; 120 kV; 20 to 100 mAs (variable depending on the patient’s weight); rotation time of 0.5 s. The images were reconstructed using a thickness of 1 mm and an inter-slice interval of 0.8 mm, using standard and high-resolution filters. Abutting axial maximum intensity projection (MIP) images 6 mm thick were reconstructed. In the patients with a history of resected non-small-cell pulmonary cancer, the CT scans were conducted after intravenous injection of an iodinated contrast agent (Iobitridol, Xenetix® 300, Guerbet, France). In the other cases, the examinations were performed without injection.
Table 1 Inclusion and exclusion criteria of the Biomarkscan protocol.

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient less than 70 years old</td>
<td>Patient more than 70 years</td>
</tr>
<tr>
<td>Living in the Grenoble area</td>
<td>Geographically distant</td>
</tr>
<tr>
<td>Accepting annual monitoring for 5 years</td>
<td>Personal reasons adversely affecting annual monitoring</td>
</tr>
<tr>
<td>At high risk of developing bronchial cancer</td>
<td>Unstable cardiac or respiratory pathology, compromising</td>
</tr>
<tr>
<td>Patient with or having a history of ENT cancer stage I and II (or III for laryngeal cancers)</td>
<td>performing the examinations and monitoring</td>
</tr>
<tr>
<td>Patient with preneoplastic bronchial lesions</td>
<td>Anticoagulant treatment or disease with a haemorrhagic risk</td>
</tr>
<tr>
<td>Patient with stage I or II bronchial cancer or with a history of operated bronchial cancer in complete remission</td>
<td>Cancer during the 5 previous years, excluding cancer of the upper airways/digestive tract, basal cell carcinoma of the skin</td>
</tr>
<tr>
<td>Symptomatic patient (respiratory symptoms) but not presenting a cancer</td>
<td>History of thoracic radiotherapy</td>
</tr>
</tbody>
</table>

Fibroscopy and histopathology

Fibroscopy was usually performed after the chest CT scan, knowing the results of the latter. White light and autofluorescence endoscopy (Olympus 4077 LIFE endoscope) was performed under local anaesthetic. Any white light focal anomaly and localised fluorescence abnormality was noted, specifying the location of the suspect area, and targeted biopsy samples were taken. Only class 3 fluorescence abnormalities were considered to be pathological. The results of the bronchial biopsies were classified into five categories: normal mucosa, squamous cell metaplasia, dysplasia (slight/moderate/severe), carcinoma in situ, invasive cancer. Only carcinomas in situ and invasive cancers were considered in our study. When a surgical specimen was available there was a comparison.

Study protocol

In the first instance, the CT scans synchronous with the diagnostic fibroscopy of the 10 patients were re-read by two radiologists working in agreement. They had been previously informed that the fiberoptic examination performed in each of these patients had shown the presence of at least one endobronchial tumour lesion, but they did not know its location or presentation. A suspect lesion was defined by the presence of at least one non-calcified endobronchial nodule or mass. The lesion parameters measured were as follows:
- mean diameter of the nodule in mm (calculated from the smallest and largest axes);
- bronchial location (according to Boyden’s classification);
- invasive character (exclusively an endobronchial tumour or crossing the bronchial wall and adjacent parenchyma);
- atelectasis;
- bronchocele.

Later, after the above analysis, the CT scans stored in our establishment’s PACS were also studied retrospectively, knowing the location of the tumours found by fibroscopy and possibly operated on, in order to establish any diagnostic delay.

Results

On inclusion in the Biomarkscan protocol, 10 patients had a history of cancer: six had a history of non small-cell lung cancer which had undergone surgery and was in remission at the time of inclusion; two had a history of ENT cancer in remission; two had a history of lung cancer and ENT cancer successively, in remission. Nine patients were smokers (active smokers or having stopped) with a mean estimated consumption of 44 pack-year (20–80). The majority of these patients consumed dark tobacco: six patients smoked exclusively dark tobacco, two patients smoked blond tobacco and one patient smoked both types (Table 2).

The results are given in Table 3. Ten intrabronchial neoplastic lesions were diagnosed from histological samples: one squamous cell carcinoma in situ (lesion 10) and nine invasive cancers (lesions 1 to 9) including five adenocarcinomas and four squamous cell carcinomas. The two bronchial trees were affected equally (50% on the right, 50% on the left).

Re-reading

On re-reading, the sensitivity of CT was 80% (lesions 1 to 8). Two lesions were not visible using CT: an invasive squamous cell carcinoma of the right superior bronchus (lesion 9) and a CIS of the superior segmental bronchus of the left lower lobe (lesion 10). As far as invasion of the bronchial walls was concerned, 37.5% of the lesions (n = 3) were solely endoluminal and 62.5% (n = 5) crossed the bronchial wall (extending beyond this to invade the adjacent pulmonary parenchyma). The mean lesion diameter was 14 mm. Lastly, three cancers were associated with a bronchocele.

Fifteen endobronchial lesions were noted on re-reading, of which eight were true positives (lesions 1 to 8) (Fig. 1) and seven false positives (in three patients). The aetiology of the false positives was a right intrapulmonary lymph node (n = 1), distal mucoid impaction (n = 2) (Fig. 2) or proximal bronchial secretion (n = 1) (Fig. 3), and postoperative changes (n = 3) (Fig. 4).
Endobronchial tumours in a campaign for early detection of bronchial cancer

Table 2  Evaluation of the smoking status of the 10 patients with endobronchial cancers.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Neoplastic history</th>
<th>Smoking</th>
<th>Type of tobacco</th>
<th>Consumption (pack-year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>55</td>
<td>Lung</td>
<td>Yes</td>
<td>Blond</td>
<td>43</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>75</td>
<td>Lung</td>
<td>Yes</td>
<td>Dark</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>70</td>
<td>Lung</td>
<td>No</td>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>62</td>
<td>Lung</td>
<td>Yes</td>
<td>Dark</td>
<td>80</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>63</td>
<td>Lung</td>
<td>Yes</td>
<td>Blond</td>
<td>27</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>53</td>
<td>ENT</td>
<td>Yes</td>
<td>Mixed</td>
<td>35</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>53</td>
<td>Lung</td>
<td>Yes</td>
<td>Dark</td>
<td>40</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>62</td>
<td>ENT</td>
<td>Yes</td>
<td>Dark</td>
<td>41</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>51</td>
<td>ENT</td>
<td>Yes</td>
<td>Dark</td>
<td>50</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>78</td>
<td>Lung</td>
<td>Yes</td>
<td>Dark</td>
<td>60</td>
</tr>
</tbody>
</table>

Table 3  Lesion parameters measured.

<table>
<thead>
<tr>
<th>n</th>
<th>Histology</th>
<th>Location</th>
<th>Visible on 1st CT reading</th>
<th>Mean diameter (mm)</th>
<th>Bronchial wall invasion</th>
<th>Associated lesion(s)</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ADC</td>
<td>LSL</td>
<td>Yes</td>
<td>11</td>
<td>No</td>
<td>Atelectasis</td>
<td>Tumourectomy</td>
</tr>
<tr>
<td>2</td>
<td>ADC</td>
<td>R external B5</td>
<td>Yes</td>
<td>28</td>
<td>Yes</td>
<td>None</td>
<td>Lobectomy</td>
</tr>
<tr>
<td>3</td>
<td>ADC</td>
<td>L B7</td>
<td>Yes</td>
<td>4</td>
<td>Yes</td>
<td>None</td>
<td>Pneumonectomy (totalisation)</td>
</tr>
<tr>
<td>4</td>
<td>ADC</td>
<td>Mid. lob.</td>
<td>No</td>
<td>6</td>
<td>No</td>
<td>None</td>
<td>Chemotherapy (surgery CI)</td>
</tr>
<tr>
<td>5</td>
<td>ADC</td>
<td>ML lobectomy stump + R B7 + R B8</td>
<td>No</td>
<td>35</td>
<td>Yes</td>
<td>None</td>
<td>Pneumonectomy (totalisation)</td>
</tr>
<tr>
<td>6</td>
<td>SCC</td>
<td>L B2</td>
<td>No</td>
<td>12</td>
<td>Yes</td>
<td>Bronchocele</td>
<td>Lobectomy</td>
</tr>
<tr>
<td>7</td>
<td>SCC</td>
<td>R internal B9</td>
<td>No</td>
<td>10</td>
<td>No</td>
<td>Bronchocele</td>
<td>Abstained (right lung only)</td>
</tr>
<tr>
<td>8</td>
<td>SCC</td>
<td>L external B10</td>
<td>No</td>
<td>6</td>
<td>Yes</td>
<td>Bronchocele</td>
<td>Segmentectomy</td>
</tr>
<tr>
<td>9</td>
<td>SCC</td>
<td>R. Sup. Lob.</td>
<td>No</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Lobectomy</td>
</tr>
<tr>
<td>10</td>
<td>CIS</td>
<td>L B6</td>
<td>No</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Cryotherapy</td>
</tr>
</tbody>
</table>

ADC: adenocarcinoma; SCC: squamous cell carcinoma; CIS: carcinoma in situ; NA: not analysable; S: solid; SS: semi-solid; NS: not solid.

Initial reading under clinical conditions

Of the 10 lesions, only 30% were detected by CT in clinical practice (lesions 1 to 3) and reported before fibroscopy took place, allowing directions to be given for histological sampling. Seven of the 10 lesions were not detected, but were discovered on fibroscopic screening. The sensitivity of CT in a clinical situation is therefore 30%.

Time between diagnostic processes

For endobronchial lesions shown by the initial CT (n = 3), the mean diagnostic length of time between the CT scan and the fibroscopy was 56 days (1—105 days) (Table 4). For the endobronchial lesions not shown in the CT but visible retrospectively (n = 5), the mean period between the first CT where the lesion was visible retrospectively on re-reading and diagnostic fibroscopy was 421 days (213—808 days). The mean diameter of the three cancers initially seen was

Table 4  Period of time between the first CT where the lesion was visible (on re-reading) and diagnostic fibroscopy.

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Detection First reading CT</th>
<th>Detection Re-reading CT</th>
<th>Delay in diagnosis (in days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Yes</td>
<td>Yes</td>
<td>62</td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Yes</td>
<td>Yes</td>
<td>105</td>
</tr>
<tr>
<td>4</td>
<td>No</td>
<td>Yes</td>
<td>213</td>
</tr>
<tr>
<td>5</td>
<td>No</td>
<td>Yes</td>
<td>259</td>
</tr>
<tr>
<td>6</td>
<td>No</td>
<td>Yes</td>
<td>686</td>
</tr>
<tr>
<td>7</td>
<td>No</td>
<td>Yes</td>
<td>808</td>
</tr>
<tr>
<td>8</td>
<td>No</td>
<td>Yes</td>
<td>350</td>
</tr>
<tr>
<td>9</td>
<td>No</td>
<td>No</td>
<td>NA</td>
</tr>
<tr>
<td>10</td>
<td>No</td>
<td>No</td>
<td>NA</td>
</tr>
</tbody>
</table>

NA: not analysable.®
Figure 1. Sixty-two-year-old male patient, operated on for cancer of the vocal cords. Successive discovery of a solid nodular adenocarcinoma of the right superior lobe operated in March 2004 and an adenocarcinoma in situ of the left superior lobe operated in June 2005; a: CT scan dated 3rd March 2004 on inclusion. The obstruction of the left B10 external subsegmental bronchus by an intrabronchial tumour was not revealed by CT or synchronous fibroscopy; b: CT scan dated 8th August 2004. No indication of bronchial obstruction; c: CT scan dated 16th June 2005 following discovery by bronchial fibroscopy of an endoluminal tumour of the left B10 bronchus which would be treated surgically (squamous cell carcinoma). The patient was alive in 2011.

Figure 2. Obstructive endobronchial lesion of the right external B4 bronchus, of \(5 \times 2\) mm. This was a distal mucoid impaction, which had disappeared on a later CT scan.

14.3 mm (4–28) and the mean diameter of the five cancers identified retrospectively was 13.8 mm (6–35).

Discussion

Our study shows that in this population with a very high risk of bronchial cancer, neoplastic intrabronchial lesions diagnosed by fibroscopy represented 37\% (10/27) of the bronchopulmonary cancers diagnosed. This proportion is high compared with other studies such as that of Feng Li who reported 83 cases of bronchopulmonary cancer in a series of 17,892 CT scans (subjects followed up for 2 years), among which five (6%) were endobronchial cancers [19]. This discrepancy may be explained by different recruitment: our study included smokers at very high risk because they had already been treated for pulmonary or ENT cancer or were symptomatic, whereas Feng Li’s patients included a majority of non-smokers (54%). Our figures should be compared with those of McWilliams et al. [18] who undertook a screening
campaign based on bimodal monitoring (CT scan and sputum analysis, then autofluorescence fibroscopy) in a population of 561 subjects who were more than 50 years old with a lower risk than our population, being only smokers (more than 30 pack-year). Twenty-eight cancers were detected in 22 patients including seven (25%) by fibroscopy and 21 (75%) by CT scan.

Loewen et al. carried out a screening study by bimodal monitoring (CT scan and fibroscopy) in a population (n = 169 subjects) at very high risk of bronchial cancer due to exposure to two of the following risk factors:
- smoking more than 20 pack-year;
- pulmonary disease related to asbestos on the X-ray;
- COPD with FEV1 less than 70;
- history of cancer of the upper airways/digestive tract without signs of recurrence after 2 years [16].

Thirteen lung cancers were discovered in 169 subjects (prevalence: 7%). Seven of these 13 cancers were stage 0 or 1a. Fibroscopy revealed five cancers out of 13 (38%) while the CT scan showed eight (62%); these figures are comparable with our study. In contrast, Kakinuma et al. reported seven patients in whom the initial screening CT scan did not detect their bronchial cancer finally shown on a later CT scan, from among 22 cancers in 1443 subjects [20]. All these nodules were peripheral. While intraparenchymatous lung cancers remain the most common and have been widely studied in the literature [2–11,21,22], intrabronchial cancers are not rare, in particular in high and very high-risk populations. This means that particular attention must be paid when reading scans, in particular when the patients smoke dark tobacco [15]. Early detection of endobronchial tumours is a prognostic advantage, since when this is the case early curative treatment can be given, either endoscopic when there is severe dysplasia or a cancer in situ, or surgical for invasive cancers [23]. In our series, cancer in situ was treated by cryotherapy and six
invasive cancers out of nine were treated by oncological surgery.

Diagnosis of proximal cancer relies above all on white light fibroscopy, which is still the gold standard [24]. This allows the airways to be inspected to detect and biopsy endobronchial lesions, and carry out bronchial brushing; it also provides the possibility of performing transbronchial punctures of hilar/mediastinal nodules [25]. Most teams use white light fibroscopy combined with the autofluorescence technique, which gives better sensitivity but is associated with lower specificity [16,17,26–28]. To date, few studies have noted the role of CT scans in the detection of endobronchial cancers. These lesions are often unrecognised or not described when interpreting chest scans, representing a not insignificant proportion of the diagnostic errors. In Aritiszbal’s study on 64 patients with an intraparenchymatous nodule monitored using CT scans and fibroscopy, 11 endobronchial tumours (17%) were diagnosed by fibroscopy but not detected in the CT scan [29]. White’s study [14] reported 14 cancers which could not be seen in the CT scan, the main characteristics of which were their endobronchial location and being in the inferior lobes. Gurney reported nine cases of bronchial cancer ignored by CT and their outcome: four cases out of nine were in a central location and the length of time between the initial examination and detection of the tumour was 3 to 14 months [30]. In our study, during the initial radiological reading, the sensitivity of the CT scan was low (30%), the tracheobronchial tree being sometimes neglected in favour of careful reading of the pulmonary parenchyma. This reading bias is aggravated by the detection of a suspect intrapulmonary abnormality, which then retains most of the radiologist’s attention. The radiologist being distracted in his reading is reported in several studies [14,19,30]. The difficulties encountered in detecting endobronchial lesions also arise from the fact that the central airways form a complex area in the CT scan. The complexity was heightened in our population in which there was a history of thoracic surgery for cancer in eight cases out of 10, resulting in false positives. Several studies have reported the difficulties of detecting focal abnormalities of the mucosa, or of differentiating endobronchial masses from extrinsic bronchial compression [31,32]. These phenomena should be alleviated with the use of isotropic volume acquisitions offering high spatial resolution (HVR), allowing multiplanar reformations and 3D reconstructions. HVR acquisitions have significantly improved the exploration of the central airways, allowing more accurate determination of the contours of the tumour, local extension and the relationship with neighbouring structures, thus adding to the fibroscopic data [33]. These detection errors can result in delayed diagnosis, which was the case in our study since the period of time between the first CT scan where the lesion was visible (retrospective analysis of the different scans for each patient on re-reading) and the diagnostic fibroscopy was 463 days.

The difficulties of detecting endobronchial cancers ought to lead to special vigilance, particularly to thorough examination of the tracheobronchial tree. Certain indirect radiological signs, although non-specific, should attract the radiologist’s attention, such as the presence of atelectasis and/or a bronchocele, which are frequently associated with endobronchial lesions (20% and 30% of the cases respectively, in our study). Images of mucoid impaction or bronchoceles are also the source of false positives; discovering them should lead to a cough and sputum test before acquiring another chest CT which may show mobilisation or disappearance of these images. In addition, the patient should be questioned to search for risk factors for endobronchial cancer, such as the consumption of dark tobacco [15]. The sensitivity of the CT scan may be considerably increased by radiological reading particularly directed towards the airways. In this way, sensitivity was increased in our study from 30% to 80%. Nevertheless, this increase in sensitivity was associated with the occurrence of false positives. In our study, post-lobectomy endobronchial inflammatory changes formed the majority of the false positives (43%). The morphology of these changes may be suspected when they have irregular contours or appear to be “budding”, causing more or less obstructive stenosis possibly associated with ventilation problems beyond. We would be deluding ourselves if we thought we could use CT to decide between inflammatory changes and a cancerous lesion. For this reason, it seems essential to note the presence of this type of lesion in the radiological report, so that an additional fibroscopic examination can be performed to obtain targeted histological samples.

**Conclusion**

In conclusion, CT screening of bronchial cancer in a population at very high risk of this disease must include careful reading of the tracheobronchial tree to avoid neglecting endobronchial tumours in the course of development and to reduce the time before therapeutic management.

**Disclosure of interest**

The authors declare that they have no conflicts of interest concerning this article.

**References**


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