

may be to use this as a covariate in case-control studies and examine whether risk is modified. This would require that cases and controls had appropriate genetic material available for analysis and would probably require a prospective study.

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Transbronchial needle aspiration in the diagnosis of mediastinal amyloidosis

We read with interest the pulmonary puzzle by Khor *et al*¹ on mediastinal lymph node amyloidosis diagnosed by direct real-time endobronchial ultrasound-guided (EBUS) transbronchial needle aspiration (TBNA). The development of EBUS-TBNA is improving the diagnostic yield of mediastinal lymphadenopathy.² However, we offer some comments about the diagnostic approach used in the case described.

First, in our opinion, conventional TBNA continues to have a significant value in diagnosing mediastinal node involvement, especially in cases of lymph nodes with a short axis >1 cm that are readily accessible.³ The potential advantages of TBNA over EBUS-TBNA are the lower cost, routine availability, ease of mastery, reduced need for patient sedation and the possibility of using histology needles that allow acquisition of a core of tissue, improving diagnostic sensitivity for other lesions such as tuberculosis, lymphoma and sarcoidosis.³ Our group reported the first case of mediastinal amyloidosis diagnosed by TBNA using a histology TBNA needle.⁴ In our opinion, according to the characteristics of the patient described and CT chest images, the safest and most cost-effective probe that was indicated in this case was TBNA using a histology needle. Second, there is a previous report in the literature of mediastinal amyloidosis diagnosis made

with EBUS-TBNA⁵ so the report was not, as the authors stated, the first published case. Finally, we agree with the authors that, although mediastinoscopy is still considered the 'gold standard' diagnostic approach for mediastinal nodal amyloidosis, conventional TBNA sampling has value and should be considered as a less costly alternative that is universal and accessible to any bronchoscopist compared with EBUS-TBNA. In fact, it should be considered the first step in the diagnostic sequence. The debate over the role of conventional TBNA in the era of EBUS remains unresolved.

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Implementing the change in National Institute for Health and Clinical Excellence guidance on airflow obstruction grading in chronic obstructive pulmonary disease

The updated National Institute for Health and Clinical Excellence (NICE) chronic obstructive pulmonary disease (COPD) guidelines¹ and the draft national strategy

for COPD² have recommended a change in the classification of airflow obstruction severity to align them with international classifications. NICE's 2004 guidelines recognised that disease severity is not the same as the severity of airflow obstruction and has recommended using other measures such as the Medical Research Council (MRC) dyspnoea scale, exacerbation frequency and multicomponent indices.³ However, UK primary care has been encouraged to code disease severity into mild, moderate and severe COPD based on lung function alone in line with NICE's 2004 guidance on airflow obstruction.

The code for COPD is thus H3; H36 is mild COPD; H37 is moderate COPD and H38 is severe COPD.

A person with COPD and an forced expiratory volume in 1 s of 42% of predicted has until now been coded as having moderate COPD; according to NICE 2010 they should now be coded as severe airflow obstruction. However, codes do not exist for mild, moderate, severe and very severe airflow obstruction. Therefore, for both patients and primary clinicians we have a communication problem and a coding problem. Clear guidance is needed on how the disease/airflow obstruction severity should be coded on primary care records without any conflicting or confusing advice.

Perhaps the answer is to abolish the codes for mild moderate and severe COPD and for new codes for airflow obstruction based on GOLD stages 1–4 to be generated. For practical purposes of classifying COPD severity, for example, for deciding the frequency of reviews, the MRC dyspnoea scale could replace H36–8 as markers of disease severity. The MRC scale is already being recorded in primary care. In future, COPD severity codes should be based on multicomponent indices, at present a suitable index for primary care has not been chosen. The NICE guidelines recommend the use of the BODE index when its component items are available, the need for the six minute walking test will make this impractical for routine use in primary care and there is insufficient evidence to approve newer indices such as the ADO⁴ and DOSE.⁵

Action is required now to address both the coding and communication issues so that the sensible advice from NICE can be implemented without causing confusion in primary care and distress to patients.

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Reversed halo sign in pulmonary zygomycosis

We read with interest the article ‘The atoll sign’ by Walsh and Robertson¹ in the November issue of *Thorax*. The authors report a case of cryptogenic organising pneumonia with the atoll sign, also called the reversed halo sign. As mentioned by the authors, this CT sign was first described in

cryptogenic organising pneumonia and was initially considered to be specific for this disease.² It was subsequently reported in a variety of pulmonary diseases, including paracoccidioidomycosis (South American blastomycosis), tuberculosis, pulmonary sarcoidosis, lymphomatoid granulomatosis, Wegener’s granulomatosis, lipoid pneumonia and pneumococcal pneumonia.^{3–5}

We would like to highlight another important cause of the reversed halo sign: invasive pulmonary fungal infections, particularly pulmonary zygomycosis (PZ) (figure 1). In immunosuppressed patients, the presence of the reversed halo sign on CT should be considered as invasive fungal disease until proven otherwise. It is an early sign that is more frequently seen in patients with PZ than invasive pulmonary aspergillosis (IPA).⁶

Early institution of high-dose antifungal therapy is associated with improved outcomes; therefore, early recognition of invasive fungal disease is important. Moreover, because the therapy for presumed fungal pneumonia in this population is often aimed at IPA due to its higher incidence and the preferred antifungal agent for IPA is voriconazole, which is not effective against PZ, it is important to differentiate between the two entities. The presence of the reversed halo sign can be used to optimise antifungal therapy to cover PZ.

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Serum 25-hydroxy vitamin D and exercise capacity in COPD

Janssens and colleagues¹ have recently reported that vitamin D deficiency is very common in patients affected by chronic obstructive pulmonary disease (COPD) and that vitamin D status correlates with lung function. In the same issue of *Thorax*, Quint and Wedzicha² discuss potential effects of vitamin D deficiency and supplementation

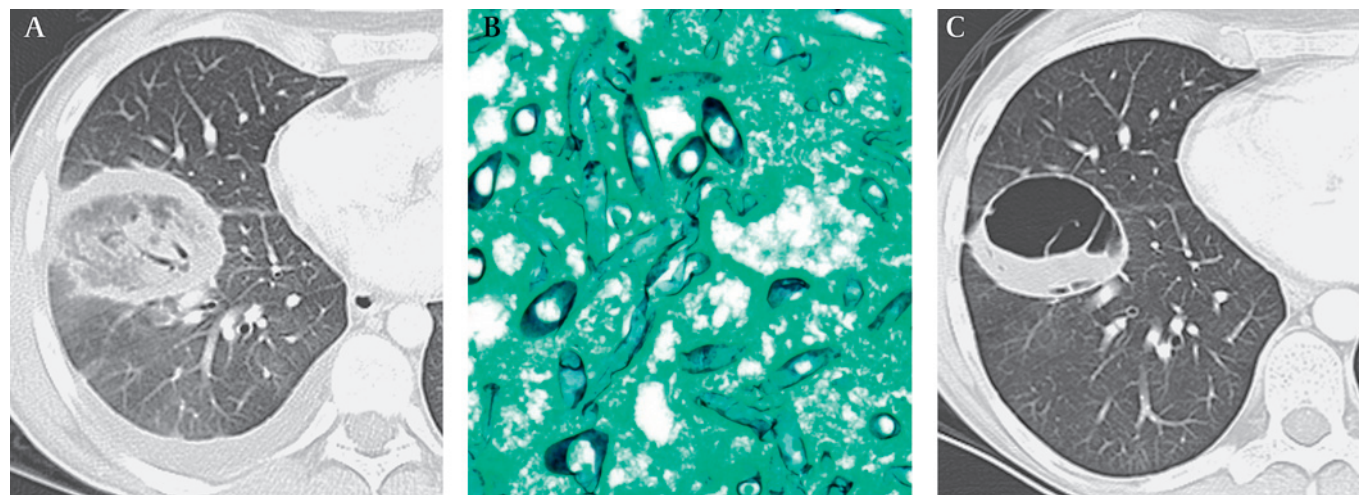


Figure 1 Pulmonary zygomycosis in a 24-year-old woman undergoing chemotherapy for recurrence of acute myelogenous leukaemia, 1 year following allogeneic stem cell transplantation, with a 2-week history of fever and dry cough. (A) Chest CT shows a focal round area of ground-glass attenuation surrounded by a ring of consolidation in the right lower lobe, consistent with the reversed halo sign. (B) Photomicrograph of the specimen from a transthoracic biopsy of the lesion shows pauciseptated hyphae with non-parallel walls and 90° branching, characteristic of *Zygomycetes* species, which is confirmed by culture. (C) One-month follow-up CT shows cavitation of the lesion, which eventually resolved under antifungal therapy.