Adiaspiromycosis mimicking widespread malignancy in a patient with pulmonary adenocarcinoma

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ABSTRACT
Adiaspiromycosis, a mycotic disease of small animals, has rarely been reported in humans. The principle causative organism in Europe is Emmonsia crescens. Inhaled, dust-borne spores of E crescens fail to germinate in host tissue, instead increasing in size dramatically to become thick-walled, round adiaspores, which induce a granulomatous response. The pathological effects depend on the spore burden and host immunocompetence, and range from asymptomatic infection through to necrogranulomatous pneumonia, respiratory failure and, rarely, death. Diagnosis is principally made through histological examination. This report describes a case of a patient with low-grade, localised pulmonary adenocarcinoma with occult adiaspiromycosis that radiologically mimicked widespread malignancy. It is believed to be the first reported human case of adiaspiromycosis in the UK.

CASE REPORT
We present a case of a 56-year-old male postal worker who was admitted to hospital following a collapse. He was a non-smoker and had no known allergies. A chest x ray revealed a right upper lobe lung tumour and bilaterally enlarged mediastinal lymph nodes. A CT of the chest showed that the tumour was next to, but not invading, a vertebral body and confirmed bilateral hilar lymphadenopathy. The patient was radiologically staged as cT2 N3 M0. Bronchoscopy was unremarkable. A CT-guided core biopsy revealed a low-grade papillary adenocarcinoma. At the multidisciplinary team discussion, it was felt that the biopsy may not be entirely representative and that the imaging indicated a more aggressive malignant process. Mediastinoscopy with lymph node biopsy was performed for treatment planning; the biopsies ruled out mediastinal nodal involvement by tumour. The patient was then referred for surgical resection. At surgery a subpleural tumour was identified in the upper lung lobe and in addition multiple subpleural nodules of up to 2 mm were noted in the upper, middle and lower lobes. There was extensive lymphadenopathy.

PATHOLOGY
Intraoperative frozen section of a wedge biopsy of one of the nodules from the right lower lobe showed focal inflammation only. The resection was performed as an upper lobectomy with systematic nodal dissection. The tumour measured 38 mm and was close to the apex of the lobe.

Microscopy of the tumour confirmed a low-grade papillary adenocarcinoma with no evidence of hilar lymph node spread, vascular invasion or pleural invasion. The patient was thus restaged as pT2 pN0. The background lung showed a significant histiocytic interstitial inflammatory process punctuated by sclerotic nodules and occasional granulomas, some with necrosis. All of the separately sampled lymph nodes were negative for malignancy but showed prominent histiocytic infiltration despite relatively little silicoanthracotic material. There were also sclerotic nodules and poorly formed granulomas with focal necrosis. No organisms were identified.

Paraffin sections from the right lower lobe wedge biopsy showed focal histiocytic infiltration and an area of scarring. A subpleural granuloma was identified and it contained a large fungal spore. The spore appeared circular in cross-section and had a thick wall with two zones: a deeply eosinophilic outer zone and a thicker, homogenous, pale inner zone. There was flocculent grey and eosinophilic material within the centre. The fungal spore was identified as an adiaspore of E crescens. No further organisms were identified in any of the tissue, despite extensive processing, indicating a low adiaspore burden.

DISCUSSION
Adiaspiromycosis, caused by Emmonsia spp, is a mycotic disease of small animals and rarely humans. While Emmonsia parva is widespread in Central Asia, Africa and the Americas, E crescens is the principal causative agent of adiaspiromycosis in Europe. Inhaled, dust-borne spores of E crescens fail to germinate in the host tissue, instead increasing in size dramatically to become thick-walled round adiaspores, which induce a granulomatous response; each granuloma typically containing a single adiaspore. A layer of epithelioid cells typically surrounds the adiaspore with foreign body and Langhan’s type giant cells often in direct contact with the spore wall. Concentric layers of fibrous connective tissue containing chronic inflammatory cells surround this. The adiaspore wall appears bilaminar on H&E staining but three zones can often be appreciated on Gomori methenamine silver and periodic acid–Schiff special stains. These are described as consisting of an inner zone with numerous laminated rings (Liesegang’s phenomenon), a thick middle zone including radial fenestrations and a thin cuticle-like outer zone. The adjacent parenchyma can show fibrosis, type II pneumocyte hyperplasia and foamy alveolar macrophages. The background lung is often minimally affected barring local...
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**REFERENCES**


**Take-home messages**

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