A 24-year-old Malay male patient was referred to the respiratory clinic because of an abnormal pre-employment chest radiograph.
He had been smoking about 10 cigarettes a day since he was 21 years of age.
He stopped smoking 10 months ago after he noticed he had being having reduced effort tolerance for the past three years.
He was an office worker and did not have a history of exposure to organic or inorganic dusts.
His two siblings were asymptomatic.
On examination, the patient was not tachypnoeic.
There were no signs of finger clubbing or pulmonary hypertension.
His oxygen saturation on room air at rest was 94% and dropped to 92% after climbing up four flights of stairs.
Spirometry testing revealed a restrictive pattern of lung disease with a forced expiratory volume in 1 second (FEV1) and a forced vital capacity (FVC) of 2.7 L (69% of predicted) and 3.2 L (68% of predicted), respectively.
The FEV1/FVC ratio was 85%.
His haemoglobin (168 g/L), serum parathyroid hormone (2.9 pmol/L [normal, 1.1-7.3]) and calcium (2.34 mmol/L) levels were normal.
24-hour urine calcium was also normal 6.9 mmol with a 24-hour urine volume of 2.8 L.
His chest radiograph (Fig.1A) revealed dense micronodular opacities distributed symmetrically and predominantly in the middle to lower zones of both lungs giving the classical "sandstorm" appearance.
The cardiac borders were obscured by the sand-like opacities.
A high-resolution computed tomography (HRCT) scan of the lungs (Fig.1B) showed widespread tiny microcalcifications throughout the lungs with a preponderance of microliths in the lower lobes.
There were associated areas of interlobular septal thickening and ground-glass changes.
Subpleural cystic changes were also seen in both lower lobes giving rise to the 'black pleura sign' (Fig.1C) (2).
No pneumothorax or pleural effusion was present.
Both the bronchial system (including the small bronchioles) and the size of the pulmonary vessels were normal.
As this was diffuse parenchymal lung disease, videoassisted thoracic surgical (VATS) lung biopsy was planned but the procedure was converted into a mini-thoracotomy because there was difficulty in manoeuvering the endostapler.
There was a moderate pneumothorax postmini-thoracotomy from which the patient fully recovered after 5 days in the ward.
The lung biopsy specimen revealed features consistent with PAM, with numerous calcospherites within the alveolar spaces (Fig.1D).
The intervening alveolar septae were congested and showed mild fibrosis with infiltrates of mainly lymphoplasmacytic cells.