No fire without smoke

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In February 2006, an 80-year-old woman, born in India, who migrated to the UK in the 1960s, was brought to our emergency department by ambulance. She had shortness of breath, which had been present for 2 months, and gradually worsened, to a point where she was breathless at rest. She arrived on a Friday evening, so we were unable to contact her general practitioner, or to access extensive medical records. She spoke little English, but her sons interpreted for her. She had symptomatically mild ischaemic heart disease, hypertension, and type 2 diabetes, and was taking isosorbide mononitrate, dipyridamole, aspirin, allopurinol, simvastatin, ferrous gluconate, furosemide, and gliclazide. She had never smoked.

The patient had no fever, but was markedly dyspnoeic, and wheezing, at rest. At the base of the right lung, we found dullness to percussion, and reduced breath sounds. We also noted a high jugular venous pressure, and peripheral oedema. Findings on blood gas analysis were: pH 7·24, PaO_2 5·5 kPa, $PaCO_2$ 9·5 kPa, HCO_3 24·4 mmol/L, and oxygen saturation 74% on F_iO_2 of 24%: the patient had type 2 respiratory failure. Chest radiography showed consolidation of the lower zone of the right lung, and changes around the right hilum which were reported as peribronchial thickening. Diagnosing pneumonia and right heart failure, we prescribed cefuroxime and furosemide, as well as nebulisers: salbutamol and ipratropium bromide. In view of the patient's acidaemia, she was transferred briefly to the intensive-care unit, where she was given non-invasive ventilation for 8 h. She became less breathless at rest, and no longer in distress. To explore possible causes of right heart failure, we requested echocardiography, which showed normal left-ventricular function, with moderate hypertrophy of the left ventricle, diastolic dysfunction, and evidence of pulmonary hypertension (estimated systolic pulmonary artery pressure 70 mm Hg; normal range ≤30 mm Hg). CT pulmonary angiography showed no large pulmonary emboli; however, we noted bilateral hilar lymphadenopathy, and a mass (diameter 5 cm) in the upper lobe of the right lung, with smaller parenchymal nodules. This mass had not been shown by radiography done a few days before; however, CT scans from 1998, on our database, showed a mass and nodules of identical size, shape, and location. We obtained previous records, which showed that a biopsy sample had been taken from the bronchus, by bronchoscopy, and a diagnosis of anthracosis had been made (figure). We deduced that the mass was a benign anthracotic nodule, and decided to monitor it by serial CT. Because of language difficulties, we were unable to obtain satisfactory results from lung function testing. We discharged the patient after 2 weeks, as she was able to go up a flight of stairs without marked distress, and felt better. We prescribed home oxygen, nifedipine, and ramipril, in addition to the patient's pre-existing drugs. When last seen 6 weeks later, she continued to feel better, and had less shortness of breath. Around a year after this hospital admission, she died of massive haemoptysis, presumably caused by pulmonary hypertension.

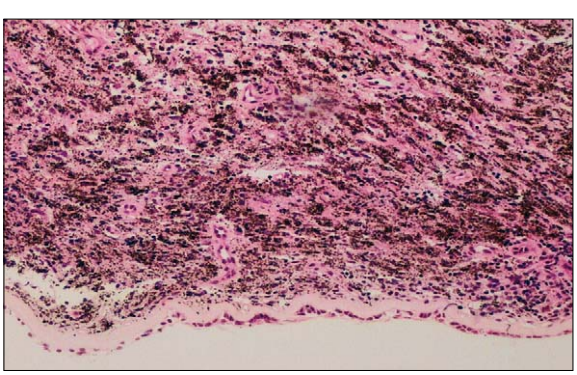

Figure: **Anthracosis**
The dark particles are from inhaled smoke.

Indian women have tended not to smoke.[1] However, many have chronic obstructive pulmonary disease (COPD) (prevalence of 1·2 to 4·5%, depending on the region).[2] More than 70% of people in India, China, and Africa cook with solid fuels, such as wood, dung, and straw and other agricultural residues.[1] If the stove is inadequately ventilated, smoke from it can damage the lungs, and increase the risk of developing COPD. Women and children are especially likely to be affected, since they spend longer near the stove.[3] Our patient had cooked indoors, using solid fuel, before migrating, and continued to do so on her annual visits to India. In 2004, indoor air pollution, from use of solid fuels, was estimated to cause 1·6 million excess deaths, and 4% of the global burden of disease.[4,5] Anthracosis—the deposition of smoke around bronchioles—can indicate exposure to indoor air pollution, but does not necessarily cause symptoms.

References

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