Pulmonary tuberculosis complicating asbestosism

YF Shea *, Janice JK Ip

Hong Kong Med J 2014;20:265.e3-5
DOI: 10.12809/hkmj134019

An 87-year-old man who previously worked in shipyard with asbestosis was admitted in November 2012 because of fever of unknown origin. He presented with fever on-and-off for 2 months and cough. On physical examination, there was no cervical lymphadenopathy or hepatosplenomegaly and the chest was clear. Complete blood picture, and liver and renal function tests remained unremarkable. Chest X-ray (CXR) and computed tomography (CT) of the thorax yielded calcified pleural plaques, diaphragmatic calcification, diffuse centrilobular nodules, and interstitial septal thickening (Fig 1). Sputum and urine cultures were negative. Further investigations included smears and cultures for acid-fast bacilli and testing for *Mycobacterium tuberculosis* (MTB) by polymerase chain reaction of sputum, urine, and bronchoalveolar larvage samples, all of which were negative. Searches for aspergillus antigen, cryptococcal antigen, the Weil-Felix test, the Widal test, nasopharyngeal aspirate for influenza and mycoplasma, urine examination for legionella antigen, autominune profiling, and tests for tumour markers, human immunodeficiency virus, and sputum cytology were all non-contributory. The patient's C-reactive protein was elevated to 3.39 mg/dL (reference range, <0.76 mg/dL). His fever had persisted on-and-off for 2 months despite multiple courses of broad-spectrum antibiotics. Positron emission tomography (PET)–CT yielded pronounced micronodules, especially over both upper lobes (Fig 1) and patchy ground glass opacities over both lower lobes with increased 18-fluorodeoxyglucose (^{18}FDG) uptake (maximum standardised uptake values of up to 4; Fig 2). The absence of pulmonary masses, mediastinal or hilar lymphadenopathy or nodular thickening of interlobular septa and any bronchovascular bundle made malignancy or lymphangitis carcinomatosis unlikely. Based on the radiology, the patient was diagnosed to have pulmonary MTB for which anti-MTB treatment was initiated. He developed sudden cardiac arrest 1 day later, and failed resuscitation. Sputum and urine sample culture results available after the patient's demise grew MTB.

Asbestosis is caused by the inhalation of asbestos which was once used as an electrical and thermal insulator. Asbestos causes fibrosis of the pleura and lung, as well as malignant mesothelioma, and PET-CT is a well-known means of picking up this complication as a linear area of intense ^{18}FDG uptake surrounding the lungs.[1] Fever of unknown origin can be due to infection, malignancy, autoimmune conditions, or drugs. Tuberculosis was one of the most common infectious causes. One of the diagnostic difficulties in our patient was the presence of interstitial lung disease making the

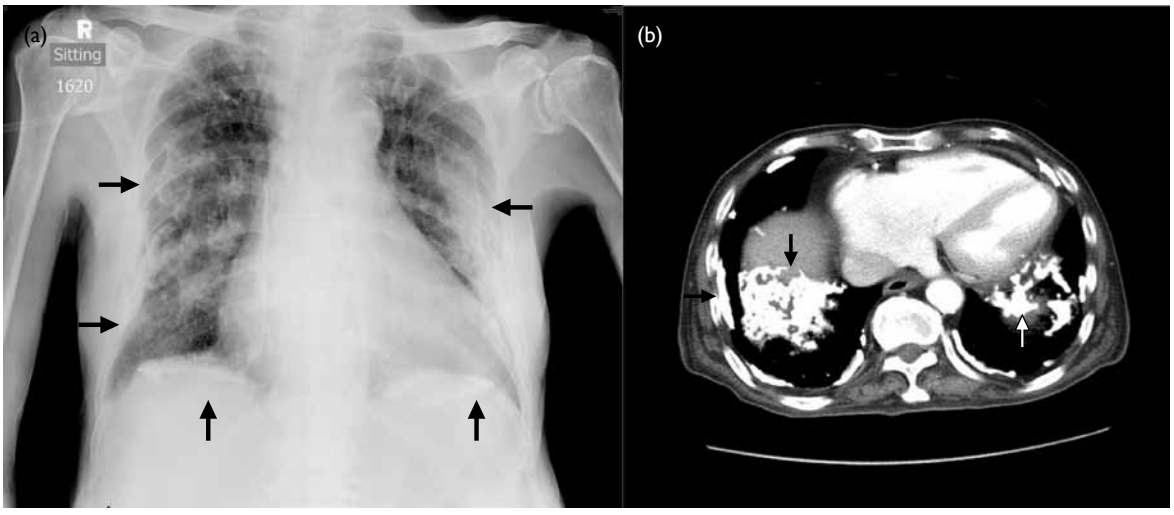

FIG 1. (a) Chest X-ray and (b) computed tomography of the thorax showing calcified pleural plaques and diaphragmatic calcifications bilaterally (arrows)

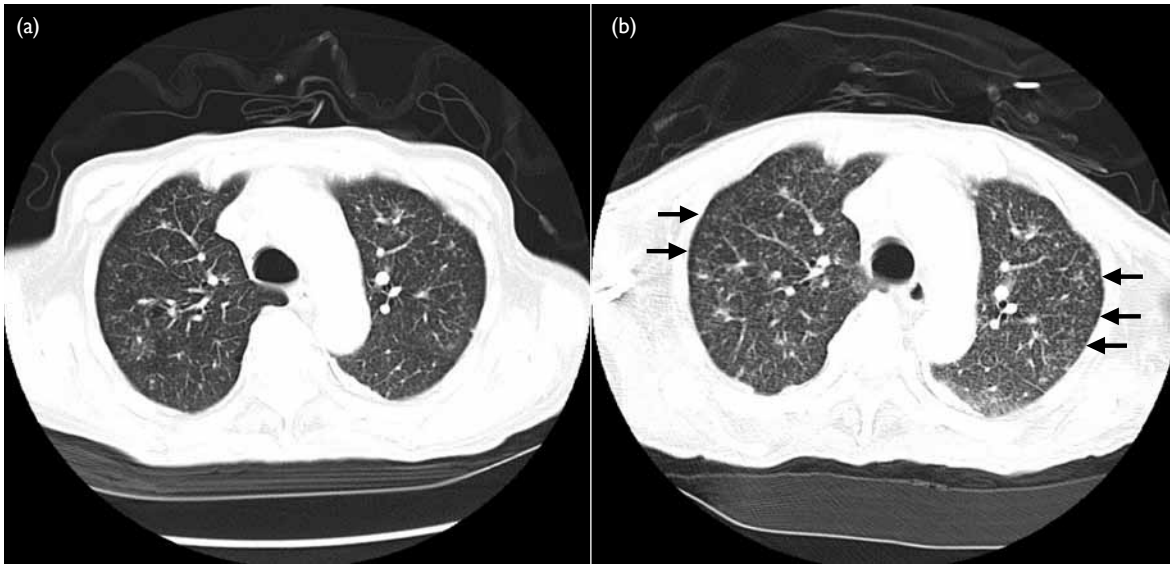

FIG 2. Computed tomography thorax (lung window) of the upper lobes taken (a) 2 months earlier and 2 months later are shown; an increase in micronodules (arrows) was evident in (b) as compared with (a)

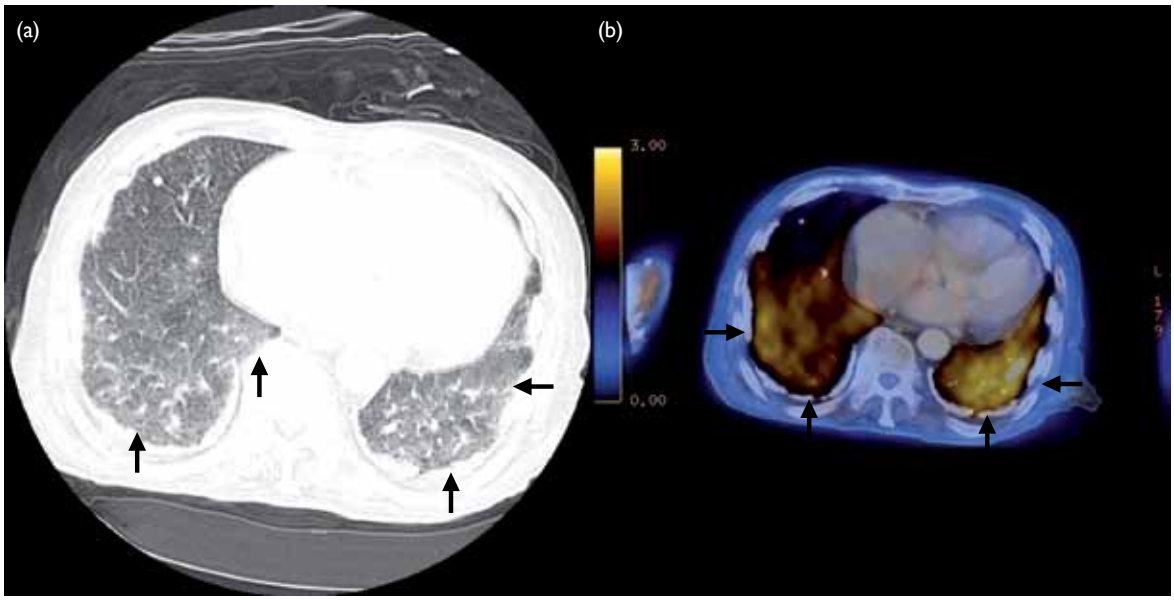

FIG 3. (a) Computed tomography thorax showing a background of calcified pleural plaques and an increase in patchy ground glass opacities over both lower lobes (arrows). (b) Positron emission tomography–computed tomography showing the parenchymal pattern of increased ^{18}fluorodeoxyglucose uptake (maximum standardised uptake values of up to 4) over the both lower lobes (arrows)

interpretation of CXR and CT images challenging. Soussan et al[2] has classified the PET appearance of pulmonary MTB into lung and lymphatic patterns and demonstrated improved diagnostic accuracy after taking account of the other specific CT changes such as upper lobe consolidation with cavitations or multiple ill-defined micronodules surrounding a cavity. Findings pertaining to our patient fitted well into previously described lung pattern of increased ^{18}FDG uptake in MTB. These included predominant lung parenchymal involvement and together with an interval excess of micronodules, especially over the both upper lobes, which led us to make a radiological diagnosis of MTB. The increased in ^{18}FDG uptake is caused by local accumulation of inflammatory cells.[3] Thus, PET-CT is useful in identifying an active

pulmonary tuberculoma in the absence of initial microbiological proof. Monitoring the response to anti-MTB treatment can also provide early evidence of drug-resistant MTB.[3,4] The earlier performance of PET-CT and institution of anti-MTB treatment may have changed the clinical outcome of our patient.

[1] **YF Shea *,** MRCP (UK), FHKAM (Medicine)
[2] **Janice JK Ip,** MB, BS, FRCR

[1] *Department of Medicine*
[2] *Department of Radiology*
Queen Mary Hospital, The University of Hong Kong, Pokfulam, Hong Kong

* Corresponding author: elphashea@gmail.com

References

1. Alavi A, Gupta N, Alberini JL, et al. Positron emission tomography imaging in nonmalignant thoracic disorders. Semin Nucl Med 2002;32:293-321.
2. Soussan M, Brillet PY, Mekinian A, et al. Patterns of pulmonary tuberculosis on FDG-PET/CT. Eur J Radiol 2012;81:2872-6.
3. Kosterink JG. Positron emission tomography in the diagnosis and treatment management of tuberculosis. Curr Pharm Des 2011;17:2875-80.
4. Demura Y, Tsuchida T, Uesaka D, et al. Usefulness of 18F-fluorodeoxyglucose positron emission tomography for diagnosing disease activity and monitoring therapeutic response in patients with pulmonary mycobacteriosis. Eur J Nucl Med Mol Imaging 2009;36:632-9.