Lipid pneumonia associated with mineral oil use presenting as fluorine-18-fluorodeoxy-D-glucose–avid lung mass

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Disclosures: Dr Cheng reports grants and contracts from Palleon Pharmaceuticals; consulting fees from AstraZeneca, Cepheid, Inivata, Janssen, Mirati, and Boehringer Ingelheim; honoraria from Potomac Center for Medical Education, WebMD, The Lynx Group; and travel support from Daiichi Sankyo, Genzyme, all outside the submitted work. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Received for publication July 11, 2022; revisions received Aug 1, 2022; accepted for publication Aug 2, 2022; available ahead of print Aug 9, 2022.

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JTCVS Techniques 2022;15:192-4

2666-2507

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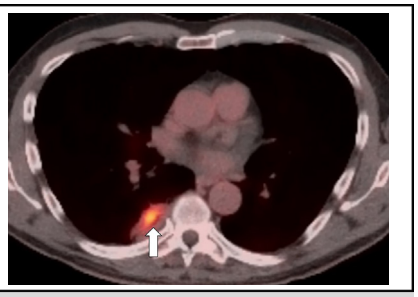
https://doi.org/10.1016/j.xjtc.2022.08.004



Intense FDG uptake at center of the lesion on PET. Pathology demonstrated lipid pneumonia.

CENTRAL MESSAGE

Lipid pneumonia may enter the differential in evaluating suspected thoracic malignancy. PET/CT characteristics may provide insight into the biology of this entity and inform diagnostic considerations.

A 57-year-old man with 120 pack-year smoking history underwent computed tomography (CT) of the chest (Figure 1), then positron-emission tomography/CT imaging (Figure 2) that demonstrated a fluorine-18-fluorodeoxy-D-glucose (FDG)-avid 4.3 × 2.2 cm consolidative right lower lobe mass (maxium standardized uptake value, 8.8), with central fat attenuation. Following nondiagnostic biopsy, video-assisted thoracoscopic wedge resection was undertaken. The Institutional Review Board of the Dana-Farber/Harvard Cancer Center approved the study protocol (No. 17-606; May 14, 2019) and publication of data. The patient provided informed written consent for the publication of the study data. Gross pathologic examination showed a subpleural yellow-tan rubbery nodule with ill-defined border (Figure 3). Histologic examination (Figure 4) showed innumerable vacuoles representing lipid material, most within the cytoplasm of macrophages and multinucleated

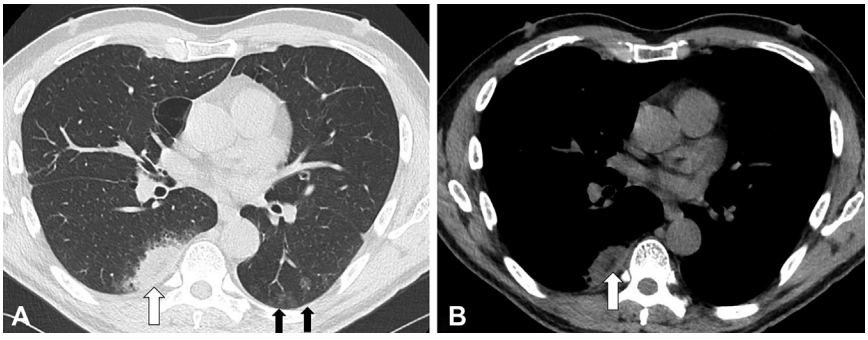


FIGURE 1. A, Axial computed tomography image in the lung window demonstrates a 4.3 × 2.2 cm consolidative mass in the posterior *right* lower lobe abutting the pleura (*white arrow*), as well as faint ground-glass opacities in the posterior *left* lower lobe (*black arrows*). B, In the mediastinal window, a central area of fat attenuation within the lesion is noted (*white arrow*).

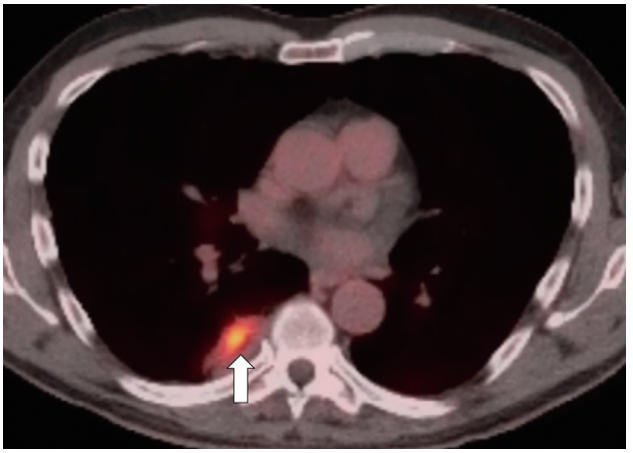


FIGURE 2. Axial fused positron-emission tomography/computed tomography image demonstrates intense fluorine-18-fluorodeoxy-D-glucose (*FDG*) uptake (maximum standardized uptake value, 8.8) at the center of the lesion corresponding to the area of fat attenuation on computed tomography image.

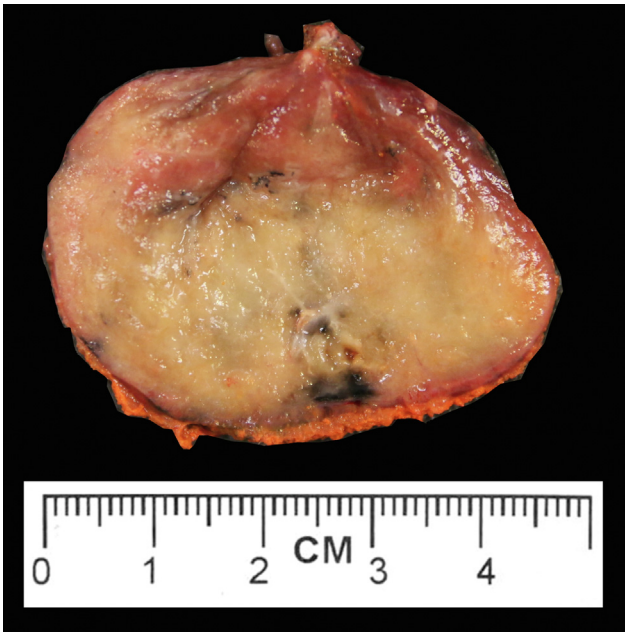


FIGURE 3. Gross pathologic examination demonstrated a subpleural *yellow*-tan rubbery nodule with an ill-defined border.

giant cells. In the center, the lipid material was incorporated into the interstitium, associated with granulation tissue, fibrosis, and chronic inflammation. At the periphery, the bulk of lipid material was located within airspace macrophages. Upon further history, the patient related longstanding use of mineral oil as a lip/face moisturizer. The positron-emission tomography/CT characteristics of exogenous lipid pneumonia[1,2] are less comprehensively described.[3,4] In this case, FDG uptake was notably strongly

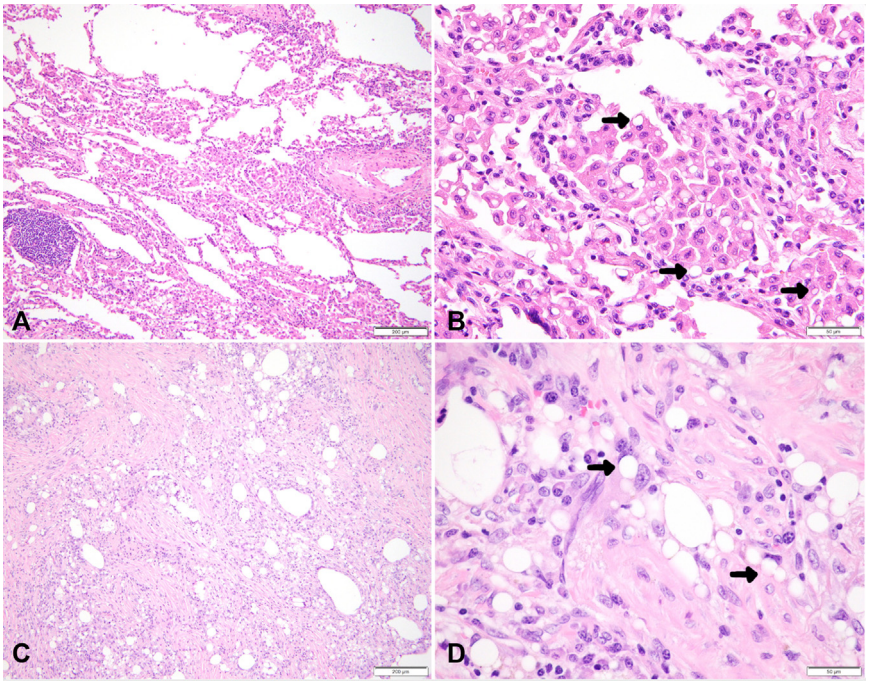


FIGURE 4. Microscopic pathologic examination demonstrated the periphery of the lesion. A, Low-power magnification shows preservation of airspaces with scattered lymphoid aggregates. B, High-power magnification shows vacuolated macrophages located primarily within the airspaces. Microscopic pathologic examination also demonstrated the center of lesion. C, Low-power magnification shows near-complete replacement of the airspaces with fibroblastic reaction. D, High-power magnification shows that the fibroblastic reaction is accompanied by neovascularization, collagen deposition, and lymphoplasmacytic inflammation with innumerable lipid vacuoles of varying sizes within the cytoplasm of macrophages and giant cells (*black arrows* indicate a few of the innumerable lipid-laden macrophages depicted.)

avid at the center of the fatty lesion. This pattern of centrally strong uptake has not, to our knowledge, been previously reported. First, it further distinguishes lipid pneumonia from cancer, which often shows a relative paucity of FDG avidity centrally due to the ischemia and necrosis that develop as tumor growth outstrips blood supply. Second, it provides insight into the biology of lipid pneumonia, supporting the pathologic finding that the exogenous lipid material organizes from the center first, with fibroblastic ingrowth and incorporation of lipids into the interstitium likely accounting for increased metabolism of glucose in the lesion. This mechanism is biologically plausible; it has not been specifically studied in lipid pneumonia but it may parallel the FDG uptake in fat-attenuating areas of atherosclerosis during inflammatory phases of disease.[5]

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